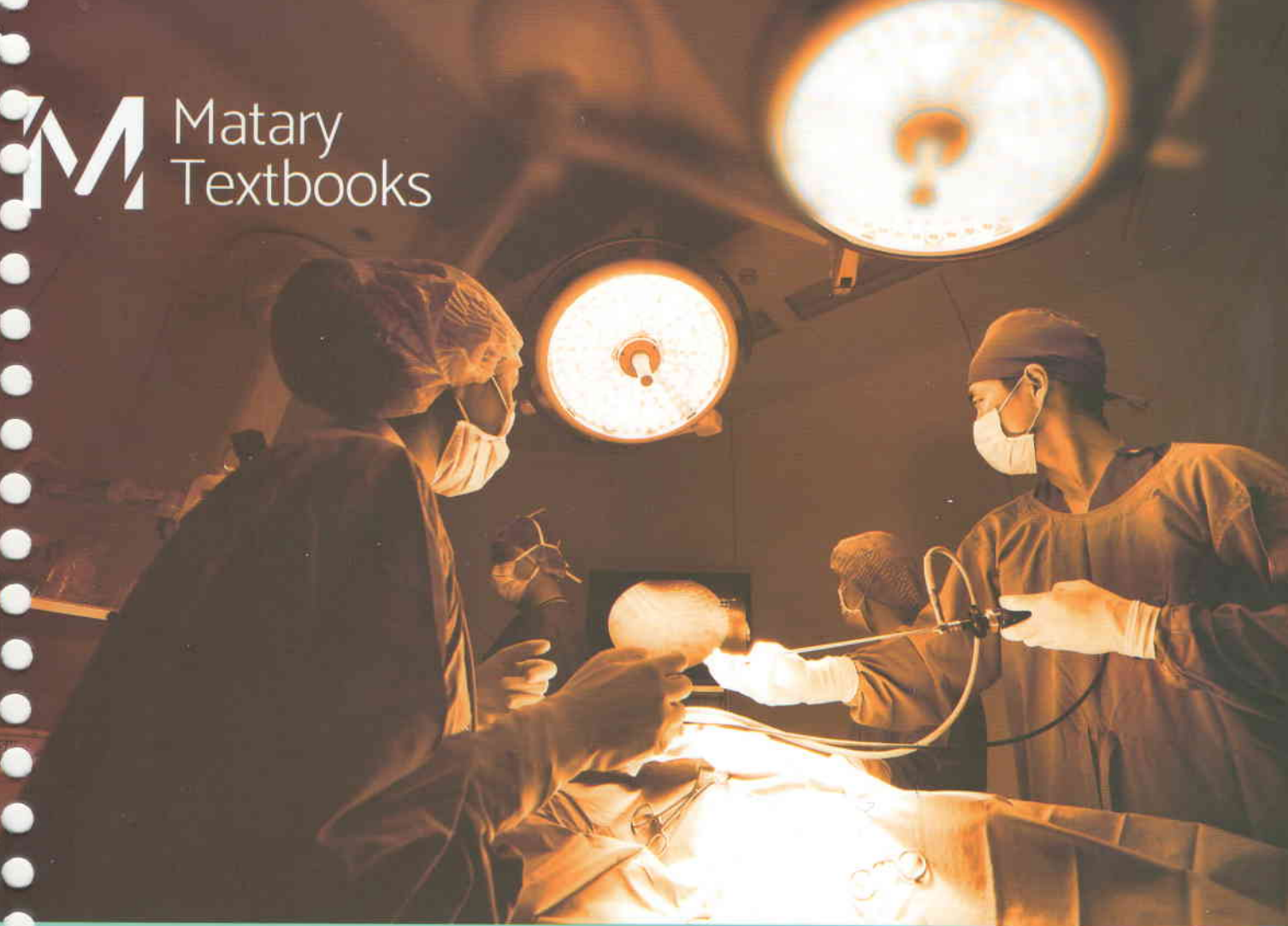




GASTROINTESTINAL SURGERY

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Caliente.COM

General Surgery

Volume III

Concise Summary of Gastrointestinal surgery



Updated

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Preface

This book provides an update for medical students who need to keep abreast of recent developments. I hope also will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of General surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you the reader will enjoy going through these pages as much as he had.

M. EI-Matary



Acknowledgement

I'd like to thank who has worked to let this
version of the book comes to light

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External abdominal hernias

Definition

Protrusion of a viscus or part of a viscus, usually within a peritoneal sac, through a defect in the abdominal wall

Etiology

I. Congenital (preformed) sac

1. Unobliterated processus vaginalis → congenital inguinal hernia
2. Unobliterated physiological umbilical hernia → congenital umbilical hernia (omphalocele)

II. Acquired cause

1. Raised intra-abdominal pressure due to :
 - a- Chronic cough
 - b- Straining at micturition or stools
 - b- Heavy work
 - d-Obesity
 - e- Abdominal swelling (splenomegaly or pregnant uterus)
2. Weak abdominal wall due to:
 - a- Obesity
 - b- Senility
 - c- Debility
 - d-Pregnancy
 - e-Weak scar
 - f- Damaged nerve supply of the muscles.

Components

1-Sac:

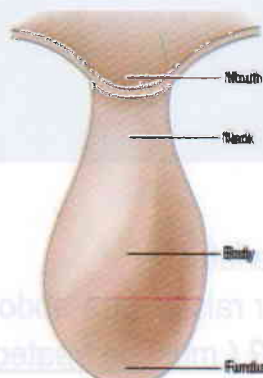
- A peritoneal pouch that bulges out through abdominal wall defect
- It has neck , body and fundus

2-Coverings:

Structures that are stretched over the sac

3-Contents: (any abdominal viscus except pancreas)

- Usually intestine (enterocele)
- omentum (omentocele) or both



-Clinical definition of hernia:

Painless swelling characterized by:

1. Reducible or give history of reducibility
2. Expansile impulse on cough
3. On anatomical site
4. With defect

- Components of hernia:

1. Sac
2. Content
3. Covering

- Neck is:

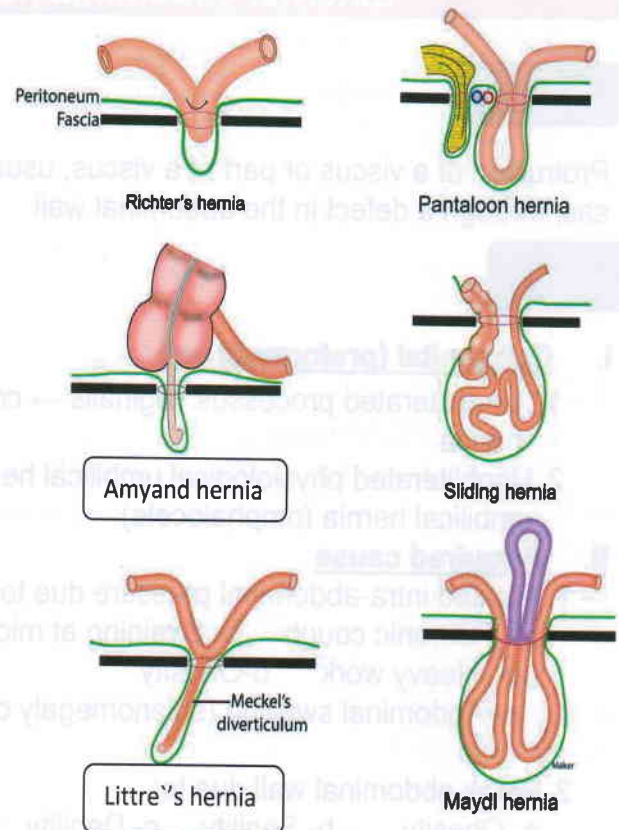
- Wide except in femoral and para-umbilical hernias
- Well defined except in incisional and direct hernias

- Reduction of a hernia means reduction of its contents into the peritoneal cavity while the sac remains empty in place

Special contents

- 1- Richter's hernia
(Common in **femoral** hernia)
The content is part of bowel circumference
Lumen is patent
- 2- Littre's hernia : the content is Meckel's diverticulum
- 3- Maydl's (W) hernia:
The content is 2 loops of intestine with intermediate loop in the peritoneal cavity
- 4- Urinary bladder
Part of the bladder may protrude into inguinal or femoral hernia
Usually protrude along inner side of the sac lying outside it forming sliding hernia

Types of Hernias



Diagnosis

These questions help diagnosis of clinically suspected hernia

- 1- Is the swelling a hernia? hernia characterized by :
 - a) Present at anatomical site
 - b) Give expansile impulse on cough (except if strangulated)
 - c) Reducible(except if irreducible ,obstructed or strangulated)
 - d) By transillumination is opaque except in children
- 2- Which type? depends on site , special features and tests
- 3- What are contents? usually intestine or omentum

	Intestine (enterocele)	Omentum (omentoceles)
Consistency	Soft	Doughy
Gurgling	Occurs during reduction	None
Ease of reduction	Difficult 1 st then easy	Easy 1 st then difficult
Percussion	Resonant	Dull

- 4- Is it complicated? see later
- 5- Is there any cause for raised intra-abdominal pressure or weak abdominal wall? (must be treated before surgery)
 - History and examination of : chest , abdomen ,urinary bladder and anus

- Weak abdominal muscles present by :
 - a) Multiplicity of hernias
 - b) Divercation of recti
 - c) Bulge of lower abdomen on straining

(Malgaigne bulge)

6- Is there any other hernia?

7- Is the patient fit for surgery?

Examine for → cardiac or respiratory problems, hypertension and diabetes

Treatment

1- In uncomplicated cases

- a) Treatment of predisposing factors first
- b) Surgery is advised to avoid complications

2- in complicated cases

- a) treatment of complication
- b) treatment of hernia
- c) treatment of predisposing factors

• there are 3 main types of hernia operations

1- Herniotomy (herniectomy) :

- Means excision of the sac
- Done alone or as part of herniorraphy or hernioplasty

2- Herniorraphy : include

- a) Herniotomy
- b) Repair of the defect by approximation of local tissue

3- Hernioplasty: include

- a) Herniotomy
- b) Closure of the defect without tension using imported material which may be:
 - I- natural as → fascia lata
 - II- synthetic mesh → polypropylene

Complications

1- Irreducibility

Definition: Failure to return the contents into the abdomen

Causes of irreducibility

1. Adhesions between the contents and the sac
2. Adhesions between the contents themselves.
3. Protrusion of more omentum within the sac.

2-Obstruction

- occurs in irreducible hernias due to occlusion of the intestinal Lumen from inside (by faecolith) or from outside (band of adhesion)

- Complications of hernia are those of the content

- Most serious complication is strangulation that needs urgent surgery

- Truss is better avoided (may precipitate strangulation) but may be used in ill, unfit patients with reducible hernia

- Polypropylene no tension repair by mesh hernioplasty is the popular nowadays and has lowest incidence of recurrence

1. irreducibility
2. obstruction
3. strangulation
4. inflammation
5. Hydrocele of hernial sac
6. Torsion of omentum

- In a purely obstructed hernia the blood supply is unaffected.

- Symptoms (of intestinal obstruction):

Vomiting, distension, colics, and absolute constipation

- Signs :

The hernia becomes distended, irreducible, but it is still **soft**

3-Inflammation (uncommon complication)

Causes

1. Rough taxis.
2. Ill-fitting truss.
3. Spontaneous inflammation of contents (appendix, fallopian tube or ovary)

Clinical picture

1. The hernia is tender but not tense
2. The overlying skin is red and edematous.

Treatment.

Operation is essential as strangulation cannot be excluded

4-Hydrocele of the hernial sac

- occurs in narrow-necked sacs if the contents return to abdomen
- The neck becomes occluded by omentum
- serous fluid collects in the sac.
- Clinical picture : cystic swelling in the upper part of the spermatic cord.
- Treatment : excision

5-Strangulated hernia (the **most serious** complication)

Definition

Constriction of contents leading to interruption of their blood supply.
If not relieved → gangrene occur within a few hours (**4-6 hours**)

Incidence

- According to the type of hernia:
 - a) about 2-4% in inguinal,
 - b) 25-30% in femoral
 - c) 15-20% in paraumbilical
 - d) 3-5% in incisional hernias.
- Strangulation can occur at any age and is commoner after prolonged use of a truss

Causes

1. Extrusion of new contents following straining.
2. Repeated attempts at reduction → producing edema.

- Irreducibility predisposes to obstruction and strangulation and so operation is essential.

- Irreducibility without other symptoms of obstruction is almost diagnostic of an Omentocele.

- Distinction between obstruction and strangulation in hernias may be difficult. It is, thus, Safer to treat it as strangulation and early surgery should be performed

- Although the incidence of strangulation is higher in femoral hernias, yet, strangulated Inguinal hernias account for more than 50% of all strangulated external hernias.(as incidence of inguinal hernia is more)

- The constricting agent may be:

1. A resistant structure outside the sac as the superficial or deep inguinal ring or the Gimbernats ligament.

2. The neck of the sac.

3. Bands of adhesions within the sac

Consequences

• If the contents are intestines:

- proximal to the strangulated loop → intestine is obstructed with progressive distension and hyperperistalsis
- Distal to the strangulated loop → intestine is collapsed.

• The strangulated loop will suffer

- Obstruction of **venous return** → congestion → accumulation of fluids → Increased congestion → hemorrhage in the wall of intestine, into lumen and *from* surface into the sac.
- Arterial** obstruction → devitalization of intestine with extrusion of content (fluid, blood and bacteria) into sac (dark toxic fluid) → gangrene

- gangrene starts at the ring of constriction then affects the Antimesenteric border (convexity) of the loop and Perforation may occur.
- Later, gangrene affects the whole loop and its mesentery.
- When infection spreads from the sac to the peritoneum → peritonitis
- Neglected cases die from septic shock and dehydration.
- Strangulated hernia may not be accompanied by intestinal obstruction if the hernia contains omentum or if it is a Richter's hernia

Clinical picture

- Severe pain at the site of the hernia.
- If the hernia contain intestine → symptoms & signs of I.O.
- Locally the hernia is tense, tender, irreducible and it does not show an expansile impulse on cough.
- In late cases, the skin overlying the hernia is red, edematous & warm.

Treatment (Urgent surgery)

I. Preoperative preparation

- Nasogastric tube suction.
- IV infusion of lactated Ringer's solution to correct hypovolaemia.
- IV broad spectrum antibiotics to guard against septicemic shock

II. Steps of the operation

- The incision should be planned to expose the fundus of the sac.
- The fundus of the sac is opened at first to evacuate the toxic fluid, which is full of organisms.
- The constricting ring should then be divided.
A grooved director or the left finger is inserted inside this ring and its coverings are divided from outside until the constriction is relieved.
- The contents are pulled out and examined, differentiate between viable and non-viable intestine at operation as below

Viable	Non viable
Normal luster	Lusterless
Pink	Grey or black
Pulsating mesenteric arteries	Not pulsating
Bleeding if injured	Does not bleed
Firm	Flabby & thin
Contracts if inched	No response

5. Dealing with the contents.

- The omentum is always excised.
 - Viable intestine is returned to the abdomen.
 - Gangrenous small intestine needs resection and primary anastomosis.
 - Gangrenous colon is treated by resection of the gangrenous segment and colostomy.
 - If the intestine looks suspicious, warm packs are applied and the patient is given pure oxygen for a few minutes. Decision is then taken whether the intestine is viable or gangrenous.
6. Repair of the hernia defect using polypropylene sutures as this suture material is inert and does not cause an inflammation
7. Subcutaneous drains are usually needed

Sliding hernia

Definition

Hernia where a viscus forms a part of the wall of the sac. The commonest are the bladder, caecum and sigmoid colon

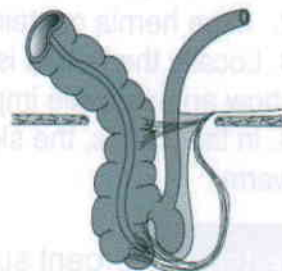
Clinical picture

- 1- Type of patient → longstanding hernia in an obese elderly man.
- 2- The hernia is usually complete oblique inguinal hernia.
- 3- the hernia is partially reducible → After reduction of the contents there is still fullness at the site of the hernia
- 4- There are urinary symptoms in case of a sliding bladder → pressing the hernia causes a desire to void, double micturition, and reduction of hernia size after micturition.

Treatment

Free the sliding sac and viscus well from the surrounding structures and push them back behind the transversalis fascia which is then repaired.

Then perform a strong repair to the inguinal canal using a mesh if required



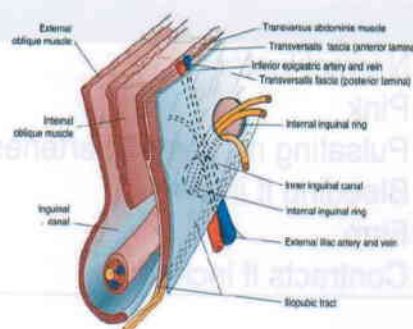
Do not try to dissect the sliding viscus from the sac as this may lead to devascularization or injury of the viscus.

Anatomy of the inguinal canal

Definition

The inguinal canal is a narrow channel about 4 cm in length at the lower part of the anterior abdominal wall.

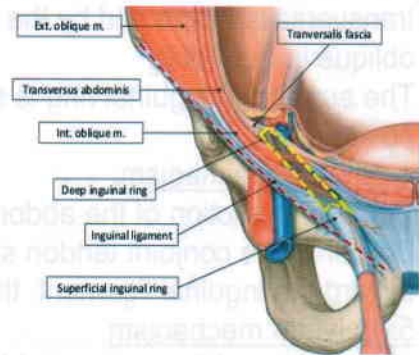
The anatomical background of the canal is to allow the passage of the testis and the spermatic cord to the scrotum



Begins

At the deep inguinal ring

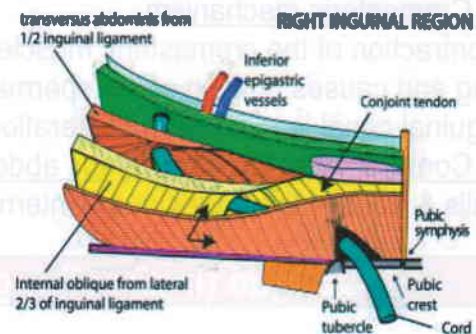
- This is an opening in the transversalis fascia 1/2 inch above the mid inguinal point (midway between the symphysis pubis and the ASIS)
- The inferior epigastric vessels run medial to it.
- The deep inguinal ring is covered anteriorly by the lower border of the internal oblique muscle.



Ends

At the superficial inguinal ring.

- This is a triangular opening in the external oblique aponeurosis that is situated half an inch above and medial to the pubic tubercle.
- It is bounded by the pubic crest below and the medial and lateral crura which are joined by intercrural fibres.
- Normally it does not admit the tip of the little finger.
- It is an exit for the spermatic cord and the ilioinguinal nerve
- It is backed by the conjoint tendon.



Direction

Medialwards with slight inclination downwards and forwards

Walls

1. The anterior wall

- the aponeurosis of the external oblique through the entire extent of the canal
- The lowest fleshy fibers of the internal oblique in the lateral half of the canal.

2. The posterior wall

- The transversalis fascia in the entire length of the Canal & conjoint tendon (in front of the fascia) in the medial half.

3. The floor

- The upper grooved surface of the inguinal ligament.

4. medially

- The lacunar ligament forms a part of the floor.

5. The roof

- the lower border of the internal oblique and the transverses abdominis muscles (arching fibers)

Protective mechanisms (to avoid development of an inguinal hernia)

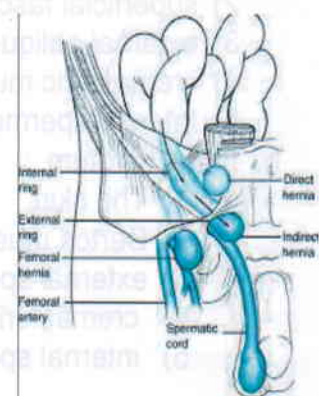
1. Obliquity of the inguinal canal.

2. The transversalis fascia

although thin, is a strong layer that supports the posterior wall

-Inguinal hernias lie above the inguinal ligament & above and medial to the pubic tubercle.

- In contrast femoral hernias' lie below the inguinal ligament & below and lateral to the pubic tubercle.



3. Weak parts of the canal are supported by strong structures.

The deep inguinal ring is reinforced by condensation of the transversalis fascia and by the fleshy fibres of lower part of internal oblique in front of it.

The superficial inguinal ring is supported by the conjoint tendon posteriorly.

4. Shutter mechanism.

During contraction of the abdominal muscles, the lower border of the conjoint tendon straightens and descends downwards toward the inguinal ligament, thus closing the posterior wall.

5. Valvular mechanism.

Contraction of the external oblique muscle tightens its aponeurosis and narrows the superficial inguinal ring.

6. Cremasteric mechanism.

Contraction of the cremasteric muscle plugs the superficial inguinal ring and causes bulging of the spermatic cord in the middle third of inguinal canal leading to its obliteration.

7. Contraction of the transversus abdominis muscle pulls & tenses the edges of the internal ring.

Transversalis fascia is a thin but strong fascial layer that lies in front of the peritoneum.

It is the most important defense against hernia formation.

-The lower part of the fascia transversalis is thickened forming the iliopubic tract which runs just above and parallel to the inguinal ligament.

-The upper part of the fascia transversalis is also thickened forming the arch of the transversus abdominis which lies at the lower border of the transversus muscle

Oblique (indirect) inguinal hernia

Etiology

- 1) Congenital (preformed) sac due to the presence of an unobliterated processus vaginalis
- 2) Acquired (pulsion) sac due to raised intra-abdominal pressure and weak abdominal wall.

Pathology

I- Defect

The stretched deep ring lateral to inferior epigastric artery

II- Sac (congenital or acquired)

Lies inside the cord coverings being anterolateral to the vas and vessels.

III- Contents usually small intestine, omentum or both

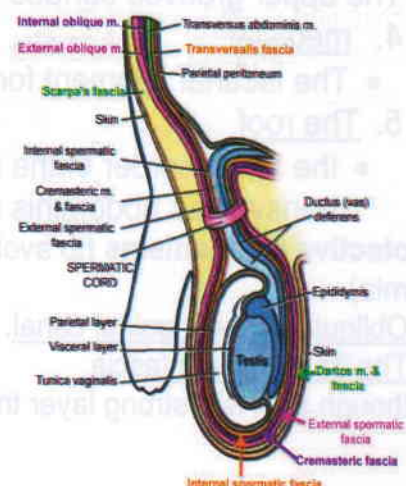
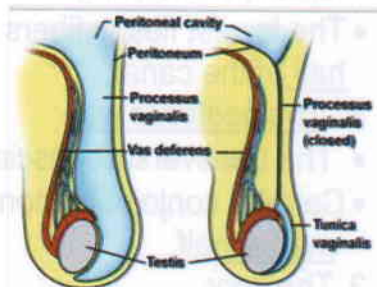
IV- Coverings.

a. In the inguinal region

- 1) The skin
- 2) superficial fascia (camper's & scarpa's)
- 3) external oblique aponeurosis
- 4) cremasteric muscle and fascia
- 5) Internal spermatic fascia.

b. In the scrotum

- 1) The skin
- 2) Dartos muscle
- 3) external spermatic fascia
- 4) cremasteric muscle and fascia
- 5) internal spermatic fascia



Anatomical types

1- Congenital type

- It is due to persistence of the whole processus vaginalis.
- The hernia reaches down to the bottom of the scrotum (Scrotal or complete hernia)
- The testis lies among the contents of the sac, the testis lies within the lower part of the hernia.
- Although called congenital, it may appear in adult life

2- Infantile type (operative finding).

- The tunica vaginalis extends upwards to the external ring, so that the sac passes down behind it.
- At operation, the tunica is liable to be opened in mistake for the true sac which will be found behind it (two sacs)

3- Adult type:

a. Bubonocele (inguinal swelling)

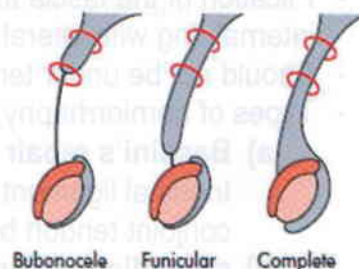
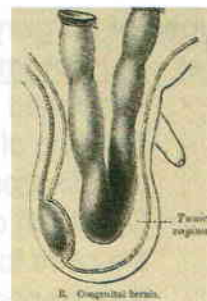
- The hernia is limited to the inguinal canal and is seen as a bulge or a swelling in the groin.
- The processus vaginalis is obliterated at the superficial inguinal ring

b. Funicular hernia

- The hernia reaches down to the neck of the scrotum.
- The processus vaginalis is closed only at its lower end, just above the epididymis.
- The tunica vaginalis is normal and the sac represents the proximal part of processus vaginalis only.

c. Complete (scrotal) hernia (inguino-scrotal swelling)

- The hernia descends to the bottom of the scrotum.
- The testis is behind the hernia and is difficult to locate



Bubonocele Funicular Complete

-Internal ring test

- Not needed if the hernia is scrotal (it is sure to be oblique)

2. Technique:

- The patient lies down & flexes the knees to relax the abdominal muscles.
- The hernia is reduced.
- The deep ring is pressed by the thumb & the patient stands up & coughs
 - Oblique hernia → does not come out except after release of the thumb
 - Direct hernia → comes out despite occluding internal ring (as it comes directly from posterior wall of inguinal canal)

Clinical picture

Symptoms

- Painless inguinal or inguino-scrotal swelling.
- Sometimes there is mild groin pain in early stages.
- The presence of severe acute pain indicates a complication.

Signs

- inguinal or inguino-scrotal oblong swelling with a narrow neck & wide fundus
- Swelling shows an expansile impulse on cough and is reducible
- Descent of contents is → downwards, forwards and medially.
- Reduction is → obliquely upwards, laterally and backwards.
- Internal ring test to differentiate oblique from direct hernia

Treatment

(By **surgery**)

Preoperative preparation

Treat any cause of increased intra-abdominal pressure as

- Difficulty in micturition
- Bronchitis
- Abdominal swellings
- ascities
- obesity.

Types of surgery

a) **Herniotomy**

- Performed in cases of hernias in infants and children.
- Opening of fundus of the sac → reduction of the content → transfixation of the proper neck → excision of the sac
- Operation can be performed through the external ring without the need to open the inguinal canal.
- Recurrence is rare and is due to failure to ligate the sac at the proper neck.
- If strangulation is neglected → testicular atrophy

b) **Herniorrhaphy**

- Indicated for hernias of adults.
- Idea: to strengthen posterior wall of the inguinal canal
- Herniotomy done first then repair of hernial defect
- Performed by nonabsorbable suture material as polypropylene (prolene).
- Plication of the fascia transversalis and reconstruction of the internal ring with lateral displacement of the cord
- Should not be under tension.
- Types of herniorrhaphy:

a) **Bassini's repair**

Inguinal ligament is sutured to the aponeurotic part of the conjoint tendon behind the spermatic cord

b) **shouldice repair**

c) **McVay's repair**

} not common now

c) **Hernioplasty**

- Obliteration of the hernia defect using tissues not from the vicinity of the hernia
- Not associated with tissue tension → has lowest recurrence rate
- Indications :
 - a) the defect is wide
 - b) The musculo-aponeurotic boundaries are too weak to hold sutures.
- Types :

a) **Onlay mesh hernioplasty.**

After excision of the sac, a mesh is placed behind the spermatic cord, and is fixed to:

Transversus abdominis and its aponeurotic arch (superiorly)

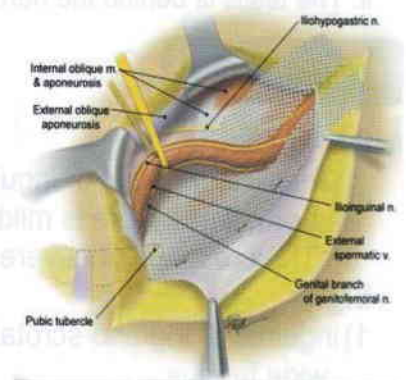
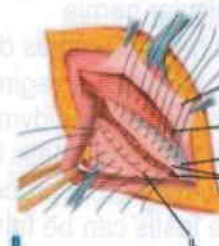
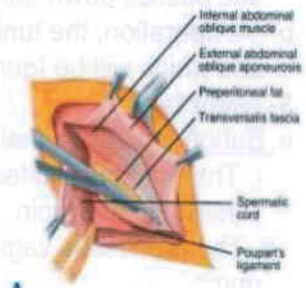
Iliopubic tract and the inguinal ligament (inferiorly)

The interstices of the mesh will be impregnated with a dense sheet of fibrous tissue. This operation called → **Lichtenstein tension free mesh repair.**

b) **Preperitoneal hernioplasty.**

The mesh is placed between the peritoneum and the fascia transversalis (can be done by open surgery or laparoscopic)

-Operation is necessary because of the risk of strangulation.
-A truss is indicated only if there is a contraindication to surgery.



Direct inguinal hernia

Incidence

- 1- common in elderly males
- 2- it may arise in young following appendectomy due to injury of the ilioinguinal nerve causing paralysis of conjoint tendon

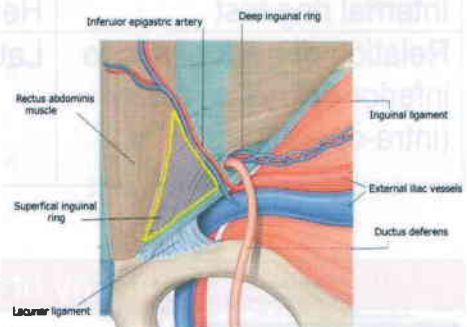
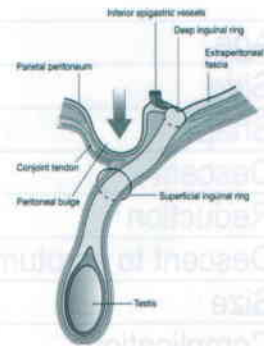
Etiology

- 1- Weakness of the lower abdominal muscles with chronic cough or straining due to urinary problems
- 2- Defect in conjoint tendon

Defect

Hasselbach's triangle, which is bounded

- medially by → lateral border of rectus muscle
- Laterally by → the inferior epigastric vessels
- Inferiorly by → the medial half of the inguinal ligament.



Clinical picture

- 1- **Type of patient** → old males.
- 2- **Symptoms**
 - painless reducible swelling
 - Bilaterality is common→ Malgaigne bulge
- 3- **Signs**
 - Inguinal swelling
 - Gives expansile impulse on cough
 - Descend → forward
 - Reducible → backward
 - Internal ring → -ve
- 4- **Complications** → are unusual



Treatment

(Essentially surgical)

1. Removal of the cause of straining.
Prostatectomy should precede hernia repair in an elderly male with senile enlargement of the prostate,.
2. Operation.
Repair of the weak posterior wall of the inguinal canal by plication of the fascia transversalis and mesh hernioplasty is indicated.

- In practice, females never develop direct hernia
-Herniotomy is not usually needed as the sac is small and is composed mainly of extra peritoneal fat
-A truss may be indicated if the patient is not fit for surgery

Difference between oblique and direct inguinal hernia

	oblique	Direct
Age	Any age	Elderly
Side	Uni or bilateral	Commonly bilateral
Shape	Oblong	Hemispherical
Descent	Downward ,forward & medially	Forwards
Reduction	Upward ,backward & laterally	backwards
Descent to scrotum	May occur	Very rare
Size	May reach large size	Usually small
Complications	Liable to occur	Rare
Internal ring test	Hernia not protrude	Hernia Protrudes
Relation of neck of sac to inferior epigastric artery (intra-operative)	Lateral to the artery	Medial to the artery

Anatomy of femoral canal

Femoral sheath

It surrounds the upper 4 cm of femoral vessels
(In the upper part of femoral triangle)

Compartments

1- Lateral compartment :

Femoral artery and femoral branch of genitofemoral nerve

2- middle compartment : femoral vein

3- medial compartment (femoral canal) : fat & LN of cloquet

Femoral canal

- The most medial compartment of the femoral sheath.
- About 1/2 inch long.

- The femoral canal is cone shaped:

a) Mouth (femoral ring) → open upwards behind the inguinal ligament

b) Apex → below and is formed by fusion of the medial border of femoral sheath and the septum between the femoral canal and the femoral vein

Relations of the femoral ring are:

- Anteriorly: Inguinal ligament

(**Poupart's ligament**).

- Posteriorly: Pectineal fascia and pectineal ligament

(**Cooper's ligament**).

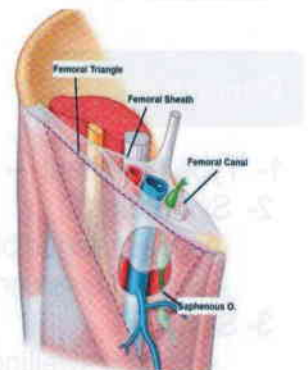
- Laterally: Femoral vein.

- Medially: Lacunar (**Gimbernat's**) ligament

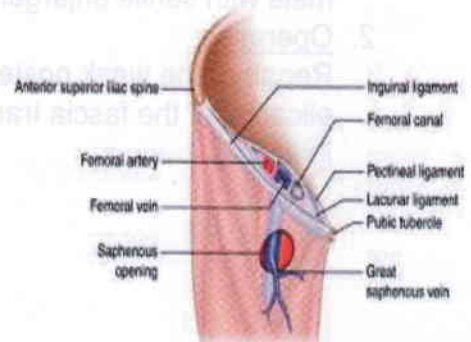
Contents: Fat, lymphatics & one LN of Cloquet

Function:

To give a space for expansion of the femoral vein



Apex of the canal is closed thus saphenous opening is found on the anterior wall of femoral sheath



Femoral hernia

Components

I- Sac

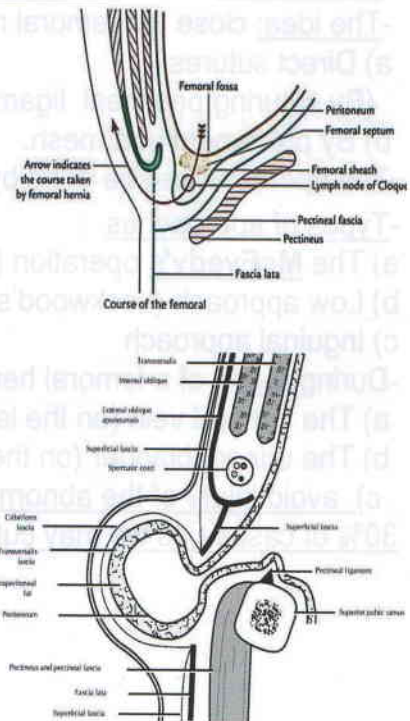
- The sac of a femoral hernia proceeds downwards in the femoral canal then forwards stretching the cribriform fascia then upwards and laterally towards inguinal ligament.
- The neck of the sac is narrow→ complications are common (irreducibility and strangulation)

II- Contents

- Usually omentum, bowel or only part of the circumference of the bowel (Richter's hernia).

III-Coverings

1. Stretched femoral septum.
2. Transversalis fascia (from anterior wall of the canal)
3. Cribriform fascia.
4. Superficial fascia.
5. Skin.



Clinical picture

- I-Type of patient→ is more common in females (Especially after repeated pregnancies)

II- Symptoms

Rounded painless swelling (unless complicated)

III- Signs

- rounded swelling with anatomical site →below the medial part of inguinal ligament, and below and lateral to the pubic tubercle
- gives an expansile impulse on cough
- direction of reduction is downwards then backwards & finally upwards
- Pressure on the saphenous opening obliterates the impulse & prevents descent of the hernia
- negative internal inguinal ring test
- may present for the first time with strangulation (40 %)

Differential diagnosis

(Swelling in femoral triangle)

Reducible	Irreducible
Inguinal hernia.	Irreducible inguinal hernia
Saphena varix	Lipoma
Aneurysm of the femoral artery	Inguinal lymphadenopathy
Psoas abscess.	Iliopsoas bursa.

-Abnormal obturator artery may be present which represents enlarged pubic branch of inferior epigastric artery when obturator artery is congenitally absent (30% of cases)

-In 10% of cases it is present in dangerous site (behind lacunar ligament as we might cut the lacunar ligament during repair of hernia)

-In 20% of cases it is present in safe site behind the symphysis pubis or lateral to the ring

- A truss is contraindicated because the possibility of strangulation is inguinal hernia because it lies high

Treatment

(**Surgery** is the only line of treatment)

-The idea: close the femoral ring either by :

a) Direct sutures

(By suturing pectineal ligament with inguinal ligament)

b) By placement of a mesh.

-The operation can be done by open surgery or by laparoscopy.

-Types of approaches :

a) The **McEvedy's** operation (the preferable) (high approach)

b) Low approach (Lockwood's)

c) Inguinal approach

-During repair of a femoral hernia the surgeon should be aware of:

a) The femoral vein (on the lateral side)

b) The urinary bladder (on the medial side)

c) avoid injury of the abnormal obturator artery which is present in 30% of cases (as we may cut lacunar ligament during repair)

-Femoral hernia is the most liable for strangulation because:

- The neck of the sac is narrow
- The constricting agent (which is the crescentic edge of Gimbernat's ligament) is sharp.

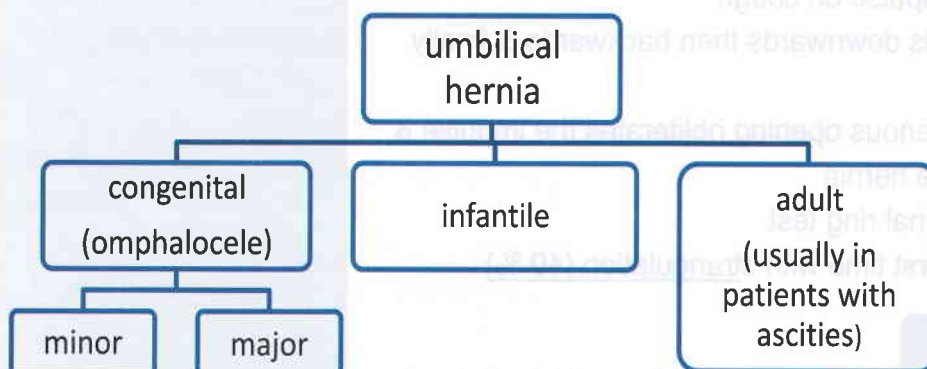
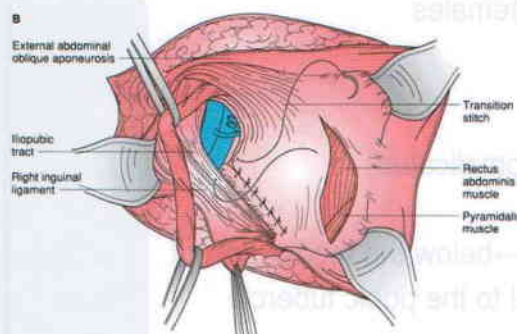
-if occurred→
present with :

- Acutely painful groin swelling
- The hernia is tense, tender, irreducible with no impulse on cough.
- +/- features of I.O

- I.O. is absent if the content of the sac is:

- Omentum
- Meckel's diverticulum (littre's hernia)
- Part of the circumference of bowel (Richter's hernia)

McVay Repair



Congenital umbilical hernia

Definition

Protrusion of viscus or part of it through congenital defect in the abdominal wall

Incidence

- Midline abdominal wall defect noted in 1:5000 live births
- Associated abnormalities occur in **30-70%** of infants and include chromosomal abnormalities and congenital heart disease.
- Fascial defects less than 1 cm in diameter close spontaneously by 5 years of age in **95%** of cases.
- When the fascial defect is greater than 1.5 cm in diameter, it seldom closes spontaneously

Indications for surgical repair of umbilical defect:

- The intestine becomes incarcerated
- Fascial defect is > 1.5 cm
- All children > 4 years of age.

	Exomphalos minor	Exomphalos major
Defect	Small (<5 cm) At the umbilicus (central cord)	Large (>5 cm) in the center of the abdominal wall, usually supra umbilical (eccentric cord)
Sac	Small (peritoneum)	Large (peritoneum)
Coverings	Layer of Wharton's jelly and a layer of amniotic membrane.	Only a layer of amniotic membrane.
Content	Usually intestine or Meckel's diverticulum.	may include many viscera and occasionally a part of the liver
Complications	During ligation of the umbilical cord , a loop of intestine may entangled in the ligature accidentally → resection (umbilico-enteric fistula)	the sac and the coverings may rupture → infection → peritonitis (the cause of death)
Treatment	-The contents are reduced and returned to the abdomen, -the sac is excised -the defect is repaired in layers	By urgent operation. <u>Usually there is no room in the abdomen to accommodate the contents.</u> o <u>If the sac is intact</u> →the defect is closed by a synthetic mesh. (orogastric tube should be placed on suction to minimize intestinal distention) o <u>If the sac has ruptured</u> : - Skin flaps are used. - The skin on either sides of the defect is undermined creating skin flaps which are brought together over the sac and sutured - Release incisions in the flanks are needed

d) After several months the peritoneum and muscles can be approximated and closed in layers.

If the viscera reduce but abdominal wall closure is not possible, there are two options:

a) A staged repair

- aims to create a protective extra-abdominal extension of the peritoneal cavity (termed a silo)

- allowing gradual reduction of the viscera and gradual abdominal wall expansion using two parallel sheets of reinforced Silastic sheeting sutured to the fascial edges or a preformed one-piece silo with a collapsible ring at its base for ease of insertion.

b) A prosthetic patch repair

- bridges the fascial gap with a synthetic material (eg, polytetrafluoroethylene) and the skin is closed over the patch

with severe associated anomalies or a giant omphalocele

- The amnion is allowed to dry and form an eschar.

- The membrane becomes vascularized beneath the eschar, and contraction of the wound with skin growth covers the defect

- A ventral hernia results, which is repaired electively when the patient is stable.

Minor omphalocele



Major omphalocele



Infantile umbilical hernia

Etiology

- 1) Weakness of the umbilical scar from infection of the umbilical cord stump
- 2) Increased intra-abdominal pressure from coughing or abdominal distension

Clinical picture

- 1) Painless swelling (unless complicated) exactly at the umbilicus
- 2) Obstruction and strangulation are rare below the age of 3 years.

Treatment

Reassurance of the parents and follow-up are the usual measures.

-The defect closes spontaneously within two years in most of the cases.

-Correction of the cause of straining, if present

- Indications for operation:

- a) The defect is more than 2 fingers wide
- b) When the hernia persists after the age of two years.

- Technique:

- a) A semicircular incision is done below the umbilicus,
- b) A skin flap is turned upwards.
- c) The sac is transfixed and excised,
- d) The defect in linea Alba is closed with few stitches of non-absorbable suture material as polypropylene (prolene).



Pathology:

1. Defect: exactly at the umbilicus
2. Sac: peritoneum
3. Contents: omentum, bowel or both
4. The coverings: extra peritoneal fat and umbilical scar
5. The neck of the sac: is wide
6. The edge of the defect: palpated as a firm ring

Adult umbilical hernia (Para-umbilical)

Incidence

More frequent in middle aged females, especially in obese multiparous women

Pathology

I- Defect

-defect lies in the linea Alba, situated above the umbilicus (because the linea Alba is thinner and broader due to stretch by stomach).

Occasionally the defect is below the umbilicus.

- It is never lateral to it (because of the rectus abdominis muscle)

-The umbilical scar lies below the swelling.

It is compressed by the hernia and looks like a crescent.

II- The sac

-has a narrow neck with a small defect in the linea Alba.

- Adhesions inside the sac are very common especially at the fundus, rendering the hernia irreducible.



Clinical picture

- 1- There is a painless swelling above the umbilicus which gives an expansile impulse on cough.
- 2- Mild dragging pain may be present in a huge hernia.
- 3- Paraumbilical hernias are frequently found to be irreducible or partially reducible.
- 4- Complications as strangulation and irreducibility are very common due to the narrow neck, sharp edge and adhesions inside the sac.
- 5- Strangulation presents with → severe acute pain

- A truss is not Satisfactory because the hernia is usually irreducible and Its use carries a high risk of strangulation.
- The previous Mayo's repair is not commonly performed nowadays

Treatment

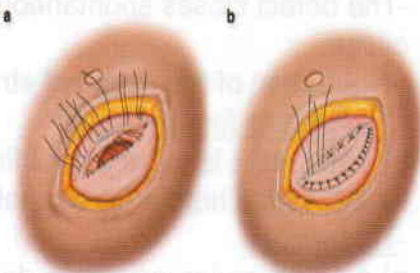
(Surgery is the only method of treatment)

-Treatment of predisposing factors prior to the surgery

Ex: For the obese reduction of weight is advised

-Technique:

- 1) An elliptical transverse incision is made over the maximum convexity of the hernia
- 2) Skin flaps are undermined upwards and downwards.
- 3) The sac is exposed and dissected down to the neck.
- 4) The sac is opened at its neck-because adhesions are usually present at the fundus.
- 5) The contents are reduced into the abdomen.
- 6) The sac is excised at the neck.
- 7) The defect is then repaired:
 - a) If the defect is the small → closed by non absorbable suture.
 - b) If the defect is large (or the musculo-aponeurotic layer is weak) → a prolene mesh is used to close the defect.



Epigastric hernia

Definition

-Protrusion of the extra peritoneal fat through a defect in the supraumbilical part of the linea Alba and is called **"fatty hernia of linea Alba"**.

-As the protrusion enlarges the fat pulls through the defect a small peritoneal pouch which may contain intestine or omentum and is called **"epigastric hernia"**



Clinical picture

I-Symptoms

- 1) May be symptomless
- 2) It may cause local pain
- 3) It may cause dyspepsia due to traction on the stomach.

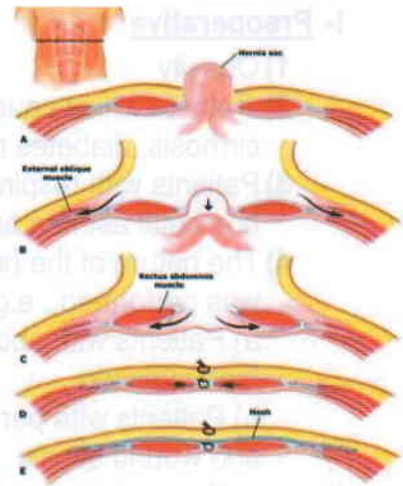
If there is pain, the surgeon should be sure that it is not due to an underlying disease, e.g. peptic ulcer or gallstones

II-Signs

- a swelling in the epigastrium which is soft, frequently irreducible and gives an expansible impulse on cough
- Occasionally there are multiple hernias.

Treatment

- 1) Treatment of predisposing factors first
- 2) operation is performed by:
 - a) excision of the protruding extra-peritoneal fat & the hernia sac
 - b) Followed by simple closure of the linea alba defect.
 - c) If the defect is large → a prolene mesh hernioplasty is performed



Divercation of recti (diastasis recti)

Definition

This is separation of the recti due to stretching of the linea Alba by a chronically raised intra-abdominal pressure.

Clinical picture

- 1) Type of patient :
 - a) in middle aged females (Due to repeated pregnancies)
 - b) in patients who have ascities and splenomegaly
- 2) When the abdomen is relaxed → nothing is visible, But on raising the shoulders → the linea Alba bulges as a longitudinal ridge and the fingers can be dipped into the abdomen between the two recti



Treatment

- 1) An abdominal belt is satisfactory in most cases.
- 2) Surgical repair is likely to fail until the cause of high intra-abdominal pressure is treated.

Incisional hernia

Definition

Is a hernia that develops at the site of a previous abdominal incision



Etiology

I- Preoperative

- 1) Obesity
- 2) Factors which cause poor healing as : malnutrition, cirrhosis, diabetes mellitus, Jaundice & corticosteroid intake
- 3) Patients with respiratory problems as: chronic bronchitis, bronchial asthma and chronic obstructive lung disease.
- 4) The nature of the primary disease for which the operation was performed, e.g.,
 - a) Patients with abdominal malignancy are usually malnourished
 - b) Patients with peritonitis will have abdominal distension and wound sepsis

II- operative

- 1) Muscle cutting incisions are more liable to burst than muscle splitting ones.
- 2) Vertical incisions have a higher incidence of burst than transverse ones.
- 3) Rough surgical technique with excessive trauma to the muscles, blood vessels and nerves.
- 4) Use of absorbable sutures in the closure of the aponeurotic layer of an abdominal wound.
Good bites should be taken on either side by non-absorbable sutures as polypropylene
- 5) Insertion of drainage tubes through the main wound.

III- Post operative

- 1) **Surgical site infection** (is the most important factor)
The tissues become friable due to collagen lysis allowing the sutures to cut through them
- 2) Poor recovery from anaesthesia leading to strong coughing
- 3) Persistent increase in intra-abdominal pressure due to repeated coughing, vomiting, hiccup or abdominal distension
- 4) Haematoma of the wound.

Treatment

Surgery offers the only definitive cure

- Many operations are available, but it is to be stressed that any repair under tension is doomed to failure.
- Usually the defect is wide and patients needs a mesh hernioplasty operation
- If the patient is unfit for surgery and provided the hernia is reducible→ an abdominal binder will keep the hernia reduced.

Recurrent hernia

-Etiology:

a) Same causes as incisional hernia

b) Specific causes:

1. Leaving a part of the original sac i.e., failure to ligate the sac at the proper neck.

2. Missing of a direct hernia sac which was present in addition to the oblique one.

3. Failure to do the proper repair e.g., doing a Bassini's repair in a patient with a weak conjoint tendon or doing the repair under tension

-Treatment:

1. Correction of any predisposing factors.
2. A truss may be applied until the patient is fit for surgery.

Hernioplasty by a synthetic mesh is usually performed

- In most of the patients with a recurrence after a repair of an oblique inguinal hernia, the recurrence will be in the medial end of the repair and will present as a direct inguinal hernia

Burst abdomen

Definition

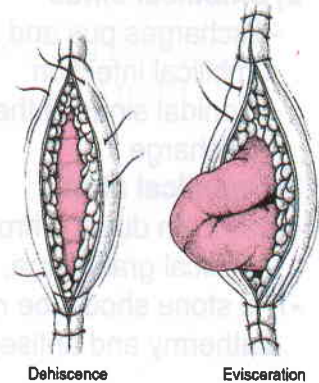
Complete disruption of an abdominal incision in the early post-operative period

Etiology

Same as incisional and recurrent hernia

Types

- a) **Partial:** The deep layers burst but the skin is intact → produces an incisional hernia.
- b) **Complete.**
 - 1- If the intestine prolapses out of the wound → called evisceration
 - 2- If the intestine is retained inside the abdomen → called dehiscence.



Clinical picture

- 1- usually occurs on **6th** to the **8th** day postoperatively
- 2- serosanguinous discharge soaking the dressing is a warning sign to the occurrence of burst and is called "**the red sign**"
- It is due to strangulation of a piece of omentum or a loop of bowel which is prolapsed through a defect in the muscles
- 3- The patient often feels as if something gives way.
- 4- Symptoms of intestinal obstruction may be present.

Treatment

By urgent surgical closure

I-preoperative

- 1) Cover the prolapsed bowel by a sterile dressing.
- 2) Insert a nasogastric tube and start an IV infusion.

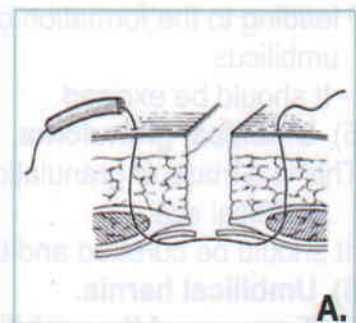
II- Operative

- 1) Under general anaesthesia, the protruding loops of bowel are washed with saline and returned to the abdominal cavity.
- 2) The omentum is spread over the intestine.
- 3) The abdominal wall is closed as one layer by **through-and-through sutures** using strong non-absorbable suture material, e.g., polypropylene → called "**retention sutures**" as they are retained for at least **3 weeks**.

Care should be taken not to puncture a loop of bowel.

III- Post operative

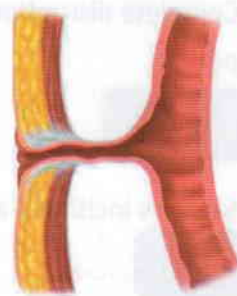
- 1) Antibiotics are prescribed
- 2) Abdominal binder is recommended.



-Burst abdomen
-Incisional hernia
-Recurrent hernia
Have common etiological factors:
1. Preoperative
2. Operative
3. Postoperative

1) Umbilical fistula

- a) Congenital from a patent vitello-intestinal duct
- b) Traumatic
- c) Inflammatory from TB of small intestine
- d) Malignant from carcinoma of the transverse colon ulcerating through the umbilicus.
- e) Urinary fistula is either:
 - i- congenital from patent urachus
 - ii- rarely acquired.
- f) Biliary fistula is very rare and may be due to operative bile duct injury during cholecystectomy.



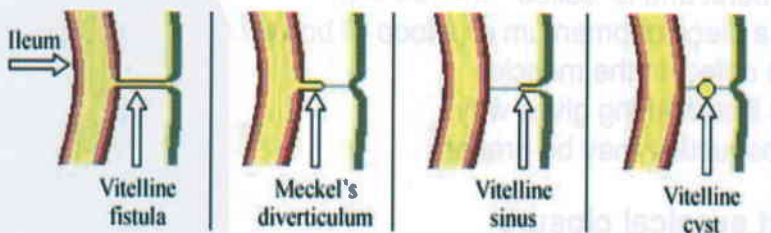
Persistent vitelline duct

2) Umbilical sinus

- discharges pus and is due to either an abdominal wall abscess or umbilical infection.
- Pilonidal sinus of the umbilicus is rare, but it leads to persistent discharge

3) Umbilical stone

- may form due to chronic inflammation of umbilicus or from umbilical granuloma.
- The stone should be removed, the granuloma is excised by diathermy and antiseptics applied.



4) Umbilical polyp

- is due to persistence of the umbilical extremity of the vitello-intestinal duct, which become everted outwards.
- The epithelial surface undergoes initiative hyperplasia from friction leading to the formation of a polypoid mass at the bottom of the umbilicus
- It should be excised.



5) Umbilical granuloma.

- This is a mass of granulation tissue due to chronic infection of the umbilical scar.
- It should be curetted and then cauterized by silver nitrate



6) Umbilical hernia.

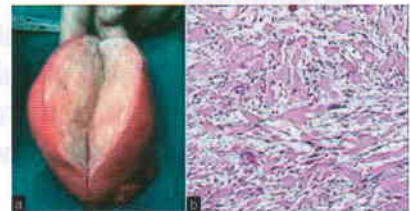
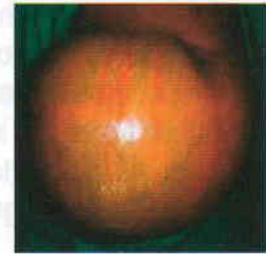
7) Tumours of the umbilicus

- a) Squamous cell carcinoma : is rare
 - It gives metastasis to axillary and inguinal LNs on both sides.
- b) 2ry carcinoma nodules may be present at the umbilicus due to spread from carcinoma of stomach, pancreas, liver or breast.

Desmoids tumor = baget's tumor = fibroma of the rectus sheath

Pathology

- 1) The nature: is not exactly known.
It is considered as a locally malignant fibrosarcoma
- 2) Arises from:
 - a- The **anterior** rectus sheath (**more common**)
 - b- The posterior rectus sheath
 - c- From the anterior abdominal wall muscles.
 - d- It may occur on top of scars or incisions.
- 3) Incidence: may be associated with intestinal polyposis (**Gardner's syndrome**).
- 4) Gross appearance :
 - A non-encapsulated, slowly growing swelling that infiltrates the surrounding structures.
 - Cut section is pinkish white.
- 5) Microscopically:
Formed of cellular fibrous tissue



Clinical picture

- 1- Type of patient : usually affects females about the age of 40 years.
- 2- Symptoms: a painless, hard, ill-defined, slowly growing mass of the abdominal wall with a nodular surface.

Treatment

- 1- By excision with a safety margin of at least **one inch**
- 2- Reconstruction of the defect by flaps of fascia or synthetic mesh.
- 3- Recurrence is very common if the tumour is not adequately excised.

Minimal access surgery

Many operations can now be performed either through the natural body orifices using fibre-optic endoscopy, or through fine stabs that are used to introduce rigid endoscopes into the peritoneum, pleura, and joint cavities

Laparoscopy

Steps

- 1) General anaesthesia
- 2) Insufflation of peritoneal cavity with CO₂ using a Veress needle (reduces the possibility of puncturing viscera during introduction).

Haematoma of the rectus sheath

-Cause :

This is usually due to trauma causing rupture of the Inferior epigastric vessels.

-Clinical picture:

there is pain, tenderness and swelling over the rectus muscle.

-Treatment:

If the haematoma is large → evacuation of the haematoma & ligation of the epigastric vessels

Gas in the peritoneal cavity makes a space between the anterior abdominal wall and the viscera → allows visualization of organs & manipulation of instruments

3. A trocar and cannula are inserted at the umbilicus.
 - The trocar is *removed* and the cannula (port) is used to introduce the telescope.
 - This telescope is connected to a video camera that displays its image on a monitor and allows the surgeon and assistants to see the interior of the abdominal cavity
4. Inspection of the peritoneal cavity.
5. Other ports are inserted under direct vision through the abdominal wall to allow the introduction of Instruments for dissection, coagulation, retraction and cutting

Advantages

1. Minimal postoperative pain.
2. Minimal impairment of pulmonary functions.
3. Fast recovery and early return to normal activities.
4. The ability to visualize and explore the whole abdominal and pelvic organs.
5. Video recording of the operative procedures with obvious educational advantages.
6. Better appearance and decreased wound problems as dehiscence or infection

Drawbacks

1. The need for well-trained surgeons.
2. High cost of the equipment.
3. Postoperative shoulder pain, which is caused by irritation and stretching of the diaphragm by CO₂.

Diagnostic laparoscopy

-Is rapidly gaining popularity in certain situations

- 1- Determination of the cause of acute lower abdominal pain e.g. acute pelvic appendicitis or torsion of an ovarian cyst.
- 2- Determination of the extent of malignant disease e.g. small liver secondaries or peritoneal nodules.
- 3- In blunt abdominal trauma to detect the exact injuries.

Frequently performed laparoscopic operations:

- 1) Cholecystectomy.
- 2) Appendicectomy
- 3) Inguinal hernia repair.
- 4) Bariatric surgery.
- 5) Fundoplication for gastro-oesophageal reflux.
- 6) Gynaecologic operations.
 - a. Tubal ligation.
 - b. Tubal adhesiolysis

-Conversion of laparoscopic surgery into the conventional open surgery is indicated in the following situations considering that the safety of the patient is the absolute priority:

1. Equipment failure.
2. Dense adhesions or anatomical abnormalities precluding safe performance of the procedure.
3. Uncontrolled bleeding.
4. Accidental injuries requiring open repair

- Diagnostic laparoscopy is only performed in stable patients

OEESOPHAGUS

Oesophageal Perforation

Etiology

1. Traumatic:

- a) Instrumental using oesophagoscopy (the commonest, **65%** of cases) during removal of foreign body or stricture dilatation

The commonest site is at the level of the cricopharyngeus muscle

- b) Foreign bodies.
- c) Swallowing of corrosives.
- d) Penetrating or blunt injuries to the neck or the chest.

2. Pathological : as in carcinoma

3. Spontaneous:

- The condition is likely to affect victims of head trauma & drunken
- In both situations there is vomiting & uncoordinated oesophageal motility
- The lower oesophagus fails to relax in front of the forcibly ejected vomitus
- Pressure markedly rises in the lower part of the oesophagus and its wall is stretched → the wall gives way either partially where the mucosa only is split producing Severe bleeding (**Mallory-Weiss syndrome**) or completely through the whole wall thickness (**Boerhaave's**)
- The tear is a longitudinal one in the lower part of the oesophagus and is usually situated in its left posterior aspect.

Clinical picture

1. Severe pain at the site of rupture.
2. The patient is acutely ill with fever, tachycardia and hypotension.
3. Mediastinal emphysema (appears as crepitus at the base of the neck, later subcutaneous emphysema over the chest wall & neck)
4. If the rupture penetrates the pleural cavity → pneumothorax or pleural effusion → respiratory distress.

Investigations

1. Plain chest x-ray:

- A) Pleural effusion B) hydropneumothorax
- C) Air in neck or mediastinum

2. Gastrografin swallow: (the best for diagnosis) → Reveal site and extent of rupture

Foreign Bodies

- Swallowed foreign bodies may be arrested in the oesophagus at points of physiological narrowing
- If the foreign body is radio-opaque it will be visualized by a plain X-ray, otherwise a dilute barium swallow will reveal it.
- Foreign bodies can be removed by the rigid or the fiberoptic oesophagoscope

Treatment

1) Cervical perforations:

- a. Nil by mouth
- b. IV hyperalimentation.
- c. Drainage of the extravasated fluid.
- d. Intensive antibiotics.
- e. If the case is early → surgical closure may be successful

2) Thoracic Perforation :

- A. If the diagnosis is early → suture of the perforation (or close the perforation by a flap from the gastric wall) and put a chest drainage.
- b. If the diagnosis is delayed → Oesophagectomy and a gastric pull up operation with chest drainage may save the patient (any attempt to close the perforation will fail)

3) Abdominal perforation: need early surgical interference

Corrosive Oesophageal Injury

Etiology

Swallowing of corrosive acids, alkalis or household bleaches

Management

- Ensure a patent airway. (the **first** to be done)
- Enquire about : a. the timing of the ingestion
b. identity of the agent c. the amount ingested
- Chest and abdominal erect x-rays are performed to detect :
 - a) any oesophageal perforation → (Pneumomediastinum)
 - b) gastric perforation → (air under the diaphragm)
- Avoid emetics or neutralization of the caustic material as may cause exothermic injury
 - Pain control
 - A course of antibiotics & corticosteroids for 4-5 days.
 - Nil by mouth until fiber optic endoscopy is performed within first 24-48 hours
 - The endoscope should be stopped at the site of severe circumferential injury to avoid perforation
- **In mild lesions:**
 - a. Patients are fed orally
 - b. Do barium oesophagogram after 3 weeks (even if there are no problems)
- **In severe lesions :**
 - a. No oral intake
 - b. NG tube is introduced under endoscopic guidance (for nutrition and as a stent)
 - c. Barium swallow after 2-3 weeks to plan definitive treatment

The common example is potash

-Pathology:

1-The extent of damage depends upon: the concentration of the chemical agent & the duration of tissue contact

2-Corrosive acids → coagulative necrosis → prevents deep penetration (less injurious than alkalis)

3-The corrosive injury → chemical burn of oropharynx, larynx, oesophagus or the stomach.

4-The burn may involve:

- a) Superficial layers of the oesophagus
- b) Whole thickness → perforation of the esophagus.

5-The injured wall of oesophagus will be replaced by scar tissue → stricture

- **later definitive treatment :**

1. **oesophageal dilatation:**

- Using bougies or balloon dilators.
- Performed every 1-2 weeks and usually 3-4 sessions
- Gastrostomy → allow retrograde dilatation (easier & safer)

2. **Surgery:**

- Indications :

- I. tight stricture with failure of dilatation
- II. dilatation is hazardous

- surgery done :

- I. first do gastrostomy (to attain an acceptable nutritional state before definitive surgery)
- II. Then colon bypass is done



Achalasia of the Oesophagus

Definition

Functional disorder of the oesophagus that is characterized by weak peristaltic waves in the body of the oesophagus and failure of relaxation of the cardiac sphincter during swallowing → functional obstruction with progressive dilatation of the oesophagus.

Clinical picture

Type of patient

- 1-more often in 2nd to 4th decades
- 2-equal in both males and females

Symptoms

1-dysphagia:

- a- painless, slowly progressive ,long standing
- b- to fluids more than solids
- c- At first intermittent but as disease progress become constant

2- **Postural regurgitation** → foul smelling

3-**pulmonary symptoms** as: aspiration, wheezing & chronic cough

Signs

General condition of the patient is usually reasonable contrary to patients with carcinoma of the oesophagus

Investigations

1- Barium swallow (**the main diagnostic tool**)

- In early cases → some hold-up of barium at the lower end of the oesophagus.
- In advanced cases → dilatation of the oesophagus & may be tortuous (**called sigmoid oesophagus**)

-The exact etiology is not known but there are theories:

- 1-Degeneration of the vagal fibres and the ganglia in the Auerbach's plexus of the oesophagus
- 2- An autoimmune disorder as there is infiltration of the oesophagus by T – lymphocytes

- In barium meal if the esophagus is interrupted above the diaphragm it is mostly carcinoma of lower end of the esophagus but if the cardia below diaphragm is narrowed it is mostly achalasia

- Smooth round termination of the lower end of the oesophagus (**Hen's or parrot beak**).
- Absence of air in the fundus of the stomach due to continuous stagnation of fluids in the oesophagus.



2- Plain chest x-ray:

- a- Fluid level in the thorax
- b- Widening of the mediastinum
- c- Aspiration pneumonitis.
- d- Absent air bubble

3- Oesophagoscopy:

- The main value is to exclude carcinoma, which may complicate 5% of cases after 20 years.
- Reveals a dilated oesophagus full of retained food and fluids.
- The cardia does not relax and may be eccentric in position (**Golf ball appearance**)



4- Manometric studies:

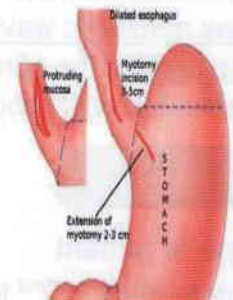
- weak peristaltic waves after deglutition
- Failure of relaxation of the cardia in response to swallowing

Treatment

1- Surgery (the most reliable method)

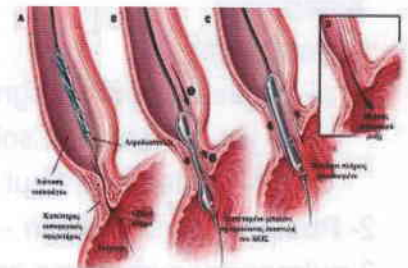
Cardiomyotomy (Heller's operation) , via a thoracic or an abdominal approach.

- expose the lower part of the oesophagus and cut the muscle fibres completely until the mucosa bulges through the incision
- not extending to cardia depending on manometry
- can be performed either by open or laparoscopic
- Advise the addition of an antireflux procedure



2- Medical treatment (of very limited clinical value)

• Nitrates or calcium channel blockers



3- Forceful dilatation of the cardia

- a- **pneumatic pressure balloons** using regiflex tube
- b- **hydrostatic balloon**

- Special balloon is introduced at the region of the cardia then inflate the balloon → rupture of the circular muscle fibres → relieve the distal oesophageal obstruction
- The oesophagus is dilated to a diameter of 3 cm and the balloon is maintained for 1-5 minutes.
- Dilatation may be complicated by hemorrhage or perforation.
- 60% of patients remain well after one year while only 30% remain symptomless after 5 years.

Recently, injection of botulinum toxin in the wall of the oesophagus at the spastic segment is tried.

Gastro-oesophageal reflux disease (GORD)

Definition

It is the reflux of gastric content into lower part of the esophagus

Incidence

- 1) The commonest upper digestive disorder
- 2) **45%** of the population having reflux symptoms.
- 3) Peptic esophagitis is the commonest endoscopic diagnosis occurring in **25%** of patients having an upper GI endoscopy

Physiology of gastro-oesophageal reflux

- 1) Alone is a physiological phenomenon which occurs after meals.
- 2) Up to 50 reflux episodes every 24 hours (most are short lived)
- 3) Exposure of the lower oesophageal mucosa to a pH < 4 should not exceed 4% of the 24 hours period, if exceed → oesophageal mucosa sensitization

Mechanisms resisting reflux

1-Competence of the cardio-oesophageal junction due to the combined action of:

- a- Lower Esophageal Sphincter (which is physiological rather than anatomical)
- b- Factors maintaining a high pressure zone of **15-25 cm H₂O**:
 - i- **the intra-abdominal esophagus** (lower 3 cm) → closed by intra-abdominal pressure
 - ii- **The action of the diaphragmatic crura** (especially the right crus) → closure of the lower oesophagus during deep inspiration or during sudden rise of the intra-abdominal pressure as in sneezing and coughing
 - iii- **The acute angle of His**: → abrupt insertion of the oesophagus into the stomach
 - iv- **The mucosal rosette** → a valve like action at the lower oesophagus.

2-Oesophageal clearance of the refluxate by 2 mechanisms

- a- **Volume clearance**: due to normal peristalsis during swallowing.
- b. **Chemical clearance**: due to neutralization by swallowed saliva (alkaline due to high bicarbonate content.)

Diffuse oesophageal spasm (Corkscrew oesophagus):

-Pathophysiology:

In this condition the normal peristaltic waves are replaced by simultaneous, Repetitive & occasionally prolonged increases in pressure in response to deglutition

-Symptoms:

- 1-Retrosternal pain (**Mainly**)
- 2-dysphagia

-Treatment:

long myotomy from the aortic arch to the cardia.



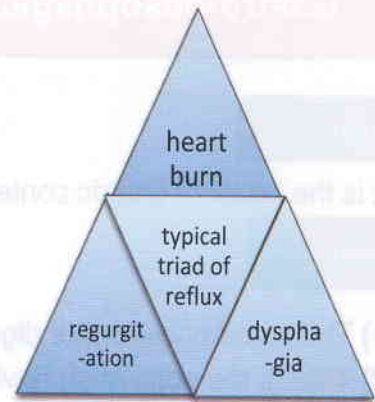
These 2 mechanisms shorten the contact time between the refluxate and the oesophageal mucosa

(1) Dysfunction of cardio- oesophageal junction:

- Primary weakness of the LOS (hypotensive LOS).
- Short length of the intra-abdominal oesophagus (hiatus hernia).
- Abnormal high number of transient lower sphincter relaxations.

(2) Gastric distention:

Impaired gastric emptying whether organic (gastric outlet obstruction) or functional (due to food which slow gastric emptying like fat, coffee and chocolate) may contribute to reflux.



Clinical picture

symptoms

A. Typical symptoms:

- | | |
|-----------------|-----------------|
| 1-heart burn | 2-regurgitation |
| 3- Dysphagia | 4-water brush |
| 5- Odynophagia. | |

B. Atypical symptoms:

- Chest pain simulating coronary artery disease.
- Pulmonary manifestations simulating bronchial asthma.
- Laryngeal manifestations as persistent by cough change in the voice or choking episodes.

Signs

- Anemia
- Haematemesis (in severe cases of esophageal erosion)

Complications

- variable degrees of erosions , ulceration and bleeding.
- strictures and shortening of the oesophagus (**schatzki ring**)
- Barrett's oesophagus:** it's a columnar metaplasia of the stratified squamous epithelium of the lower oesophagus (Metaplasia may be gastric or intestinal, Intestinal can develop into adenocarcinoma)

Investigations

1- pH study: (the best investigation)

- If the timing of these reflux symptoms coincides with a low pH recording , thus symptoms are due to reflux.

2- Oesophageal manometry:

To test the pressure of the lower oesophageal sphincter.

-Fat dyspepsia is more common in GERD than gall stone diseases
- Symptoms are aggravated by posture (lying flat, bending & stooping) ,can be severe especially at night & after large meals

- PH study:

- A small probe is passed through the nose to be positioned 5 cm above the LOS.
- The probe is connected to a digital recorder worn on a belt for 24 hours.
- The collected pH changes are then analyzed using a computer system.
- The patient is asked to record when he gets the symptoms of reflux

3- Upper GI endoscopy:

- It can evaluate the degree of esophagitis
- diagnose the presence of hiatus hernia
- a biopsy can be taken if suspicion of Barrett's oesophagus or malignancy is present.

Endoscopic grading of reflux :

- I. Grade I hyperemic mucosa
- II. Grade II intermittent superficial ulcers
- III. Grade III extensive ulceration
- IV. Grade IV stricture or Barrett's esophagus

A-Conservative: (Main line)

Life style modifications

1. Weight reduction (markedly improve the symptoms).
2. Stopping of smoking.
3. Avoidance of large, fatty, spicy and acidic meals, alcohol, chocolate and coffee.
4. Avoidance of recumbence after meals for at least 2 hours.
5. Elevation of the head and bed 15 degree to reduce reflux.

Medications

- | | |
|--------------------------|-------------------------------------|
| 1-Antacids | 2-H ₂ receptors blockers |
| 3-proton pump inhibitors | 4-prokinetic drugs. |

B-Surgical:

(10-15% will be referred to have anti reflux surgery)

Indications

- 1- Failure of medical treatment.
- 2- Development of complications as:
 - a) severe esophagitis
 - b) persistent anemia
 - c) Oesophageal stricture.
- 3- Atypical symptoms (respiratory, pharyngeal & dental).
- 4- Non compliance of the patient

Procedure (Anti reflux surgery)

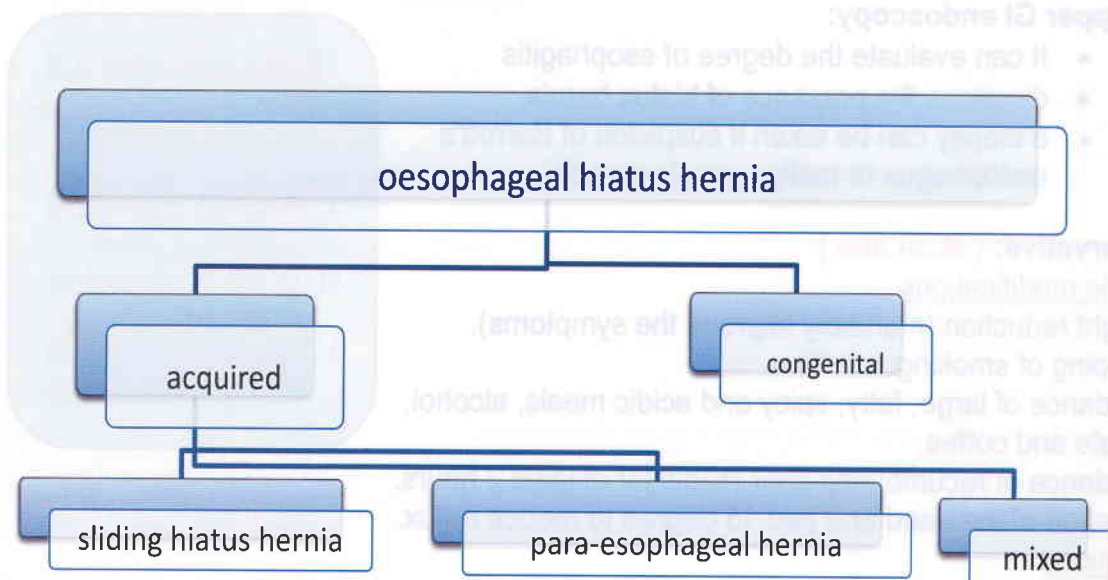
Nissen fundoplication

- includes a complete wrap of the fundus of the stomach around the lower oesophagus (360 °) to create a high pressure zone
- This operation can be done by open or laparoscopic surgery



Barrett's esophagus (10% of cases of GERD)

- Metaplasia of lower end of the esophageal mucosa into columnar type
- Metaplasia may be gastric or intestinal type ,Intestinal type is precancerous resulting in adenocarcinoma
- Regular endoscopic monitoring with multiple biopsies is essential
- Endoscopic mucosa resection using photodynamic therapy or argon beam coagulation can be used before progress to cancer
- The best line of treatment is large dose of PPI



Sliding hiatus hernia

Definition

The cardiac orifice & adjoining stomach part herniate through the oesophageal hiatus in the diaphragm into the posterior mediastinum

Etiology

- 1-Increased intra-abdominal pressure (as in pregnancy, obesity or wearing of tight corsets).
- 2-Decreased elasticity of the right crus (as in obesity and old age)

Clinical picture

Type of patient : more after the age of 40 years and it is more common in females

Symptoms

1. Commonly asymptomatic.
2. If the episodes of reflux increase → clinical picture of GORD

Investigations

1. Barium meal in the Trendlenburg's position
 - a) The hernia appears as a small epiphrenic bulge (reducible in erect position)
 - b) Widening of the oesophageal hiatus.
 - c) Reflux of barium from the stomach to oesophagus
 - d) If there is esophagitis→, irregularity of the oesophageal lumen with granular mucosal pattern
2. Perform the investigations of GORD

Pathology:

- Cardiac orifice herniate through the oesophageal hiatus → a small empty peritoneal sac on LT side of the stomach
 -The oesophagogastric junction is displaced into chest → reflux of acid gastric juice

Effect on esophageal mucosa

Esophagitis → ulceration→ metaplasia into columnar cells (Barett's esophagus) "precancerous "

Effect on muscularis mucosa

A)Circular layer→ spasm →fibrosis →stricture

B) Longitudinal layer →spasm →pull stomach up → more herniation (vicious circle)

Treatment

- sliding hiatus alone does not need treatment
- only indication for surgery → severe symptoms or complications
- principles of the operation:
 - 1- Reduction of the hernia.
 - 2- Maintains of a segment of the oesophagus intra-abdominally.
 - 3- Performance of an anti reflux mechanism (**Nissen's fundoplication**)
 - 4- Repair of the right crus of the diaphragm.

-Presence of hiatus hernia on a barium meal study does not necessarily mean that the symptoms of the patient are due to it,
 -Hiatus hernia may occur without reflux and there may be reflux without a demonstrable hiatus hernia

Para oesophageal (rolling) hernia:

Definition

- True herniation of the stomach or part of it through esophageal hiatus in front of the oesophagus in the posterior mediastinum.
- The oesophagogastric junction lies below the diaphragm → no reflux oesophagitis

Clinical picture

Compression of

- 1- Esophagus → Intermittent dysphagia.
- 2- Heart → a) cardiac symptoms as palpitation
b) Post prandial pain (confused with angina.)
3. Phrenic nerve → hiccough.

Complications

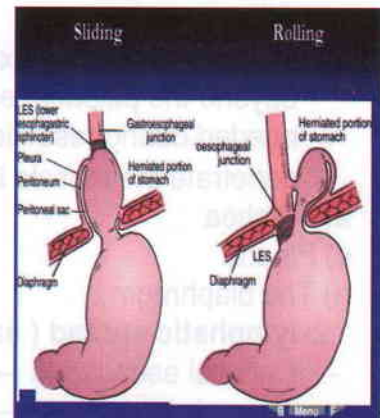
Strangulation of the herniated stomach → pressure necrosis or rupture → Mediastinitis or empyema.

Investigations

- 1- **Plain chest X-ray**: may show the gastric gas shadow in the chest.
- 2- **Barium meal**: → a) The herniated stomach
b) The oesophagogastric junction is in its normal location

Treatment

- **Surgical** to avoid fatal complications.
- Via Abdominal approach:
- Steps: a. stomach is retracted downwards
a. the hernia sac is excised
b. the defect is closed



-CT scan of the chest and upper abdomen should be done before a planned surgical repair.
 - The pain occurs after meals due to distension of the intrathoracic stomach.

Carcinoma of the oesophagus

Pathology

Site recently lower 1/3 > upper 2/3

Macroscopic

- a) An annular schirrous lesion.
- b) A malignant ulcer with raised everted edges.
- c) A fungating cauliflower-like friable mass

Microscopic

- I. Adenocarcinoma (in the lower third of the oesophagus) → on top of Barrett's oesophagus
- II. squamous cell carcinoma (is the **commonest** in the middle third)

Spread

1- Direct spread

- Spreads in circumferential and longitudinal directions of the wall of the oesophagus.
- Malignant cells can extend in the submucosa many centimeters beyond the palpable edge of the growth → so large safety margin needed during resection
- penetrate the muscle layer and infiltrate adjacent structures as:
 - a) Trachea
 - b) Pericardium
 - c) Pleura
 - d) Recurrent laryngeal nerves
 - e) The diaphragm
 - f) Stomach

2- lymphatic spread (early)

- Cervical esophagus → deep cervical lymph nodes
- Thoracic esophagus → posterior Mediastinal , tracheal , tracheobronchial LNs
- Abdominal esophagus → left gastric and celiac LNs

3- blood spread (late) to lung ,liver ,ribs or vertebrae

Clinical picture

Type of patient : male >50 years old

Symptoms :

- 1- **Dysphagia** (the **main** symptom)
- 2- **Regurgitation** → leading to pulmonary problems
- 3- **Severe and progressive loss of weight.**
- 4- **Haematemesis** (not a common symptom)
- 5- **symptoms due to infiltration of adjacent structures**
 - a) change of voice (recurrent laryngeal nerve)
 - b) recurrent choking due tracheo-oesophageal fistula

signs

- a) **general** : cachexia ,dehydration ,chest infection ,and jaundice
- b) **local** : 1.Neck → for lymph nodes enlargement or mass
 - 2. Chest → pleural effusion
 - 3. Abdomen → hepatomegaly & ascities

-Incidence:

- a) **Age** → after 50 years old
- b) **Sex** → more in **males** (except in the cervical oesophagus which is more common in females)

-Predisposing Factors:

- 1. Barrett's oesophagus (the **most important**)
- 2. Chronic irritation (by certain diets as spicy food, alcohol intake & smoking)
- 3. Corrosive stricture of the oesophagus.
- 4. Achalasia.
- 5. Long standing reflux oesophagitis.

- Characters of dysphagia

- 1. Progressive continuous
- 2. At first for solids then for soft diet and lastly for fluids.
- 3. In advanced cases the patient can't swallow his own saliva → continuous drooping of saliva → aspiration pneumonitis
- 4. By the time the patient complains of dysphagia= about 2/3 of the circumference of the oesophagus are involved by the tumour

Investigations

For diagnosis

1- Barium swallow:

- Cauliflower like lesions → persistent irregular filling defects
- Schirrous lesions → an irregular narrowed segment with overhanging margins (**Shouldering or apple core**)
- Peristaltic waves may be absent above the lesion due to submucosal infiltration of the oesophagus
- Differentiate between
 - a) Carcinoma → mild proximal dilatation with irregular termination (**Rat tail**)
 - b) Achalasia → severe proximal dilatation with regular termination (**Parrot peak**)



2- Oesophagoscopy → demonstrate the lesion & take multiple biopsies

For staging

3- Bronchoscope (in lesions of the upper oesophagus) → detect infiltration of the trachea or bronchi.

(Done **before** barium swallow because if there is fistula → barium pass into trachea)

4- Plain chest X-ray → detect pulmonary deposits or malignant effusion.

5- Abdominal ultrasound → reveal liver deposits or ascities.

6- Endoluminal ultrasonography → invasion of the mediastinum & LN enlargement.

7- CT scan → demonstrate the extent of the lesion, presence of lymph deposits & presence of infiltration of adjacent structures.

8- PET scan → assess nodal and distant metastasis

Indications for palliative treatment:

1. Patients with a poor cardio-respiratory status
2. Patients with distant metastases
3. Patients with advanced local spread as
 - a) Tracheo-oesophageal fistula
 - b) Recurrent laryngeal paralysis
 - c) Infiltration of the pleura or pericardium
 - d) Extensive LN deposits

Treatment

I. Inoperable tumors (advanced cases) 60 %

- Treatment is palliative
- Aim: relieve dysphagia and allow the patient to swallow
- Methods of palliation:

1- Intubation (the **best** method)

- insert a rigid tube through stenosed segment → patent lumen

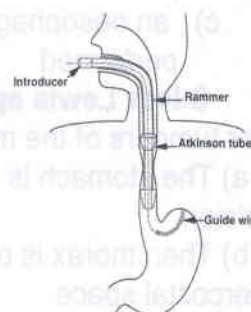
- Methods of insertion:

a) Tubes which are pushed through the lesion by the oesophagoscope (**souttar tube**)

b) Tubes which are pulled from below after doing gastrostomy (**Celestin tube**)

2-Radiotherapy (suitable for carcinomas of the upper oesophagus)

- Dose of 4000 to 4500 rads over a period of 4 weeks.
- Complications: hemorrhage , perforation and pneumonitis



There are now self expanding tubes

3-Laser photocoagulation (Nd: YAG)

- Laser energy causes tissue coagulative necrosis.
- Complications include chest pain and possible perforation.

4-Gastrostomy

- This is performed when there is no other alternative.
- It does not relieve the patient from the inability to swallow his own saliva.

II- operable tumors 40 %

- Treatment is radical surgery
- Aim is to cure the patient
- **Indications.**
 - a) Patients in a good general condition
 - b) A localized resectable without distant metastasis

• The idea

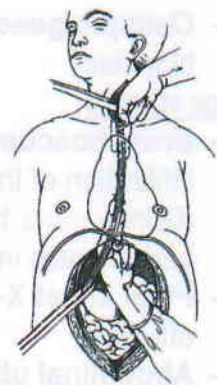
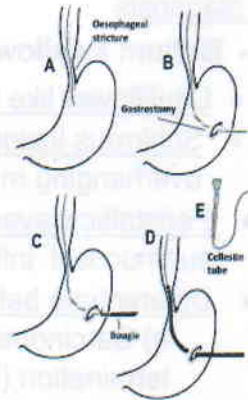
A) to resect the lesion with an adequate safety margin on either side (10 cm)

B) restore the continuity of the gastrointestinal tract

• Types of operations

1- A transhiatal oesophagectomy (the preferred surgery nowadays)

- Thoracotomy is avoided by mobilizing the esophagus from the abdomen via the diaphragmatic hiatus and via neck incision then oesophago-gastric anastomosis is performed in the neck.



Advantages:

- a) Guarantee of an adequate safety margin.
- b) The stomach can be mobilized easily.
- c) Anastomosis is in the neck, if leakage occurs → does not lead to mediastinitis or empyema.
- d) No need for a thoracotomy

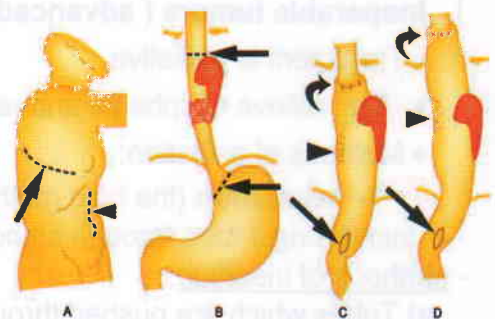
2-For tumours of the lower third of the oesophagus

- a) Do a left thoraco-abdominal incision
- b) Then excise the oesophagus and the upper portion of the stomach
- c) an oesophagogastric anastomosis is performed

3-Ivor Lewis approach

(For tumours of the middle third,) **not widely used**

- a) The stomach is mobilized through a midline incision
- b) Then thorax is opened through the right 5th intercostal space
- c) Oesophagus is excised down to the cardia.
- d) Then an oesophagogastric anastomosis is performed



Oesophageal Replacement Surgery

1. Gastric pull-up

- Idea → mobilize the stomach completely into the neck with preservation of the right gastric and right gastroepiploic vessels. .
- **Advantages of gastric replacement:**
 - a. The stomach has a very rich blood supply.
 - b. Complete serosal covering.
 - c. Easy mobilization.

2. Colon replacement.

- A segment of the right or left colon is mobilized with preservation of the feeding artery and vein and the marginal artery near the wall of the colon.
- The colon is anastomized below to the stomach and above to the cervical oesophagus

3. Pectoralis major myocutaneous flap

- can be used to replace a localized segment of the cervical oesophagus
- The skin segment is rolled upon itself to form a tube with the epidermal surface facing inwards

4. Free jejunal replacement with microvascular anastomosis.

- A segment of jejunum is separated with preservation of the feeding artery and vein.
- Using microvascular techniques the feeding artery is anastomosed to the inferior thyroid artery in the neck and the feeding vein is anastomosed to one of the veins in the neck.

Dysphagia

-Definition: Difficulty in swallowing.

-Causes:

I) In the mouth

- 1) Stomatitis
- 2) Glossitis
- 3) Neoplasms of the tongue and cheek

II) In the pharynx

A) Mechanical

1. Pharyngitis.
2. Retropharyngeal abscess.
3. Plummer Vinson syndrome
4. Pharyngeal diverticulum.
5. Post-cricoid carcinoma

B) Neuromuscular

- 1- D.M
- 2- Myopathy
- 3- Poliomyelitis.

In the oesophagus

A) Mechanical

-In the lumen: foreign bodies.

-In the wall:

1. Congenital stenosis
2. Traumatic as corrosive or post-operative stricture.
3. Inflammatory as in reflux oesophagitis.
4. Neoplastic as carcinoma.

- Compression from outside:

- 1-Malignant thyroid
- 2-malignant lymph nodes
- 3-aortic aneurysm
- 4-mediastinal tumors

B) Neuromuscular :

1. Achalasia of the cardia.
2. Paralysis of the glossopharyngeal or vagus nerves.
3. Tetanus.
4. Myasthenia gravis.
5. Hysteria

-Investigations:

- 1- Chest x-ray.
- 2-Barium swallow.
- 3- Pharyngoscopy & oesophagoscopy.
4. C.T scan of the chest

N.B: Odynophagia means painful swallowing

Therapeutic uses of oesophago-gastroduodenoscopy:

1. Band ligation or sclerotherapy of oesophageal varices.
2. Control of bleeding from peptic ulcers by submucous adrenaline injections, laser or hemo-clips. Bleeding superficial gastric erosions can be controlled by the argon beam coagulator.
3. Percutaneous endoscopic gastrostomy (PEG).
4. Dilatation of benign oesophageal strictures and/or achalasia as well as dilatation of pyloric obstruction. .
5. Stenting of malignant strictures of the oesophagus and trachea-oesophageal fistulae.
6. Recently, areas of oesophageal dysplasia or Barrett's oesophagus can be ablated by endoscopic mucosal resection (EMR) or photodynamic therapy.
7. Removal of swallowed foreign bodies.
8. ERCP with removal of CBD stones, dilatation or stenting of biliary strictures

Motility:

- The process of receptive relaxation is complimentary to the storage function in which the body & fundus of the stomach relax to accommodate increasing food volume without pressure increase.
- Gastric motility is a function of the intrinsic nerve plexus, but is modified by the autonomic innervation and the content of food:
 - The parasympathetic system → encourages gastric emptying. Division of the 2 vagal trunks → induce gastric stasis and inhibit receptive relaxation.
 - The sympathetic system → inhibits motility.
 - Solid, fatty & hyperosmolar foods → retard gastric emptying

Hydrochloric acid secretion: (in response to eating in 3 phases)

1. Cephalic phase

- mediated by → vagal stimulation that leads to:
 - a) Direct effect on parietal cells → acid secretion.
 - b) Induces gastrin release from the G cells of the antrum.
- provoked by → the sight, smell or thought of food.

2. Gastric phase

- Mediated by → the hormone gastrin (the **most powerful** stimulator of gastric acid secretion)
- Food & products of early protein digestion → mechanical distension of the antrum → gastrin release from the antral mucosa into the blood stream
- Low pH < 2 (high acidity) → inhibit gastrin release (-ve feedback)
- Gastrin act on the parietal cells through the release of histamine.

3. Intestinal phase (the least contribution)

- The involved factors are poorly understood.

Gastroduodenal mucosal barrier:

Protect the mucosa from being digested by acid & pepsin, include:

1. The mucus secreted by the mucosa → a protective lining.
2. Bicarbonate secretion by the mucosa (present between it & the protecting layer made of mucous) → the surface pH is thus higher than the luminal pH.
3. Rapid regeneration of mucosal cells.
4. Abundant mucosal blood flow.
5. Prostaglandins which maintain the blood supply.

Factors that weaken the gastroduodenal mucosal barrier:

1. Drugs

- Especially aspirin & NSAID drugs → anti-prostaglandin effect.

2. Duodenogastric reflux of bile.

3. Helicobacter pylori infection:

- secrete :a) Urease enzyme → liberation of ammonia → ↑ gastrin
- b) Proteases → destroy the mucus.

Functions of the stomach:

1. Grinding & mixing of food into a homogenous chyme.
2. Food reservoir → allows slow passage of chyme into the duodenum.
3. Early stages of protein digestion by pepsin.
4. Secretion of hydrochloric acid which:
 - a) Activates the proteolytic enzyme pepsin.
 - b) Kills bacteria in swallowed food.
 - c) Facilitates iron absorption.
5. Secretion of intrinsic factor → essential for vitamin B12 absorption

- A balance is present between:

- 1) The attacking forces (acid & pepsin)
 - 2) The defense mechanisms (mucosal barrier)
- A rise in acid secretion or weakness of the barrier → start peptic ulceration

Acute gastric dilatation

Definition

The stomach loses its tone and rapidly dilates to enormous size, sometimes filling the abdominal cavity to an extent that other viscera are totally obscured.

Pathogenesis

- The stomach becomes filled with air and dark & foul fluid
- The source of the fluid is mainly from the intravascular compartment (which is depleted & the patient passes into hypovolaemic shock)

Etiology

- 1- can occur postoperatively especially after:
 - a) pelvic operations
 - b) splenectomy
 - c) Cholecystectomy
 - d) gastric surgery (rare)
- 2- during labour (few cases)
- 3- In patients immobilized in plaster casts.
- 4- In patients suffering from debilitating or toxemic states as a terminal event.

Clinical picture

A- Early cases:

- 1- Hiccough
 - 2- Upper abdominal discomfort.
 - 3- Tachycardia.
- should be diagnosed at this stage and do gastric decompression by a nasogastric tube

B-Neglected cases:

1. Effortless vomiting of dark foul smelling fluid.
2. Picture of hypovolaemic shock.
3. Upper abdominal distension.
4. Succession splash.
5. Aspiration pneumonia may occur

Prevention

Placement of a nasogastric tube after major abdominal surgery

Treatment

- 1- Insertion of a nasogastric tube and continuous aspiration.
- 2- Attention to fluid, electrolytes and acid-base balance.

-The basic pathologic features cannot be easily explained but can often be prevented

- There is a lot of similarity between acute gastric dilatation and paralytic ileus.
 1. Both occur postoperatively.
 2. Caused by GI atony.
 3. Preventable.
 4. Vomiting and distension.
 5. Same treatment (Drip & suck = IV fluid drip to correct deficits & provide needs and nasogastric suction)



Acute peptic ulcer

Types

1- Multiple erosions.

- Site: in the body and fundus of the stomach
- Cause: in patients receiving NSAID
(Being anti-prostaglandins → weaken the mucosal barrier)
- Number & shape: They are multiple, shallow & punched out.
- Size: from 1 mm (erosions) to 1-cm or more
- Depth: usually limited to the mucosa and submucosa
- Outcome: after healing there is no evidence of scarring.
- Clinically:
 - Present as indigestion but difficult to recognize at this stage.
 - only recognized when they bleed as it need urgent upper endoscopy



2- True stress ulcers.

- Pathology: multiple erosions that if not promptly recognized and treated → coalesce to become → acute haemorrhagic gastritis → severe and intractable upper gastro-intestinal haemorrhage.
- Causes: a) ICU patients b) severe trauma
 c) Major burns (**Curling's ulcer**) d) severe acute pancreatitis
 e) Shock (specially septic shock)
- Pathogenesis: is uncertain but they represent as failure of The gastric mucosal barrier either due to:
 - a) a vascular injury with Ischaemia of the mucosa
 - b) Reflux of bile into the stomach → H^+ ion back-diffuse the mucous membrane.

Investigations

By endoscopy

Barium meal is contraindicated



Treatment

Prophylactic treatment:

- Patients at high risk of developing stress ulcers should receive either proton pump inhibitors or H₂ blockers.
- Intra luminal antacids and sucralfate may be used.

Actual treatment:

1. Discontinuation of the offending drug, if any.
2. Correction of the shock state.
3. PPIs as omeprazole or H₂ blocking agents (given orally or IV according to the severity)
4. Intraluminal antacid → maintain intraluminal pH of at least 5
5. Mucosal protectors as sucralfate.
6. Severe bleeding requires :
 - a) control of blood loss by endoscopy either →
 - i- adrenaline injection in submucosa
 - ii- LASER coagulation
 - b) Blood transfusion
7. If all previous measures fail → urgent gastrectomy

Peptic ulcer disease:

-Nature:

A PU is caused by acid-pepsin digestion of mucous membranes & only occur when there is a functioning gastric mucosa capable of secreting both acid & pepsin.

-Sites:

An acid-pepsin secreting mucosa does not digest itself but will do so to adjacent mucous membranes of the following sites:

1. **Duodenum** (commonest)
2. The alkaline gastric antrum
3. The jejunum (after a gastrojejunostomy)
4. The lower oesophagus
5. The small intestines adjacent to Mickle's diverticulum containing ectopic gastric mucosa (rare)

Chronic duodenal ulcer

Etiology

Multiple factors, it is associated with increased gastric acidity

1. *Helicobacter pylori*

- found in the mucosa of **85%** of duodenal & **75%** of gastric ulcer
- produces urease enzyme → transforms urea to ammonia → alkalinity of the antrum → increased production of gastrin

2. NSAIDs (Non-steroidal anti-inflammatory drugs):

- They inhibit prostaglandin → decrease mucosal blood flow
- They inhibit proliferation of mucosal cells → preventing healing

3. Diseases associated with increased gastrin production:

a. Zollinger Ellison syndrome (rare condition) either by :

- A pancreatic tumour secretes excessive gastrin hormone
- Hyperplasia of G cells in the antrum (less common)

b. Hyperparathyroidism: (unusual cause)

- High calcium level stimulates excessive gastric production.

4. Genetic predisposition:

- Acts by producing a large parietal cell mass (double the normal)

5. An increased vagal tone (maximum at night)

- **evidenced** by increased amount of overnight gastric acid secretion.

Pathology

Gross appearance:

- Size: usually small.
- Site: - in the 1st part of the duodenum
 - on the posterior wall (more common)
 - On the anterior wall (less common)
 - Sometimes 2 ulcers occur (one on the anterior & the other on the posterior wall) → called "**kissing ulcers**"
- Consistency: Induration.
 - Repeated healing attempts → excessive fibrosis → gross duodenal deformity (seen at operation & in serial duodenal radiographs)
- Edge: sloping edge
- Floor: covered by a thin layer of granulation tissue.
- Base: usually lies in the muscle coat or may even extend to lie in the pancreas.

Complications:

- 1- Perforation (in anterior ulcers)
- 2- Bleeding (in posterior ulcers because of their close proximity to the gastroduodenal artery or its branches)
- 3- Pyloric (gastric outlet) obstruction due to dense fibrosis

Clinical picture

Type of patient:

In young active persons (appear well fed & plump)

- Eradication of *H. pylori* from the stomach reduces the recurrence rate following medical treatment

- The high gastrin level → marked elevation of HCL acid production

- Smoking will most probably not cause an ulcer, but it may delay its healing.

- An active ulcer is associated with marked induration surrounding it → mistaken for malignant disease



Symptoms:

1. Pain (the leading symptom)

a. Site : deep seated in the epigastrium

b. Type : often described as boring.

c. Time : a few hours after meals (**hunger pain**).

Undiluted gastric acid pass to the duodenum → irritates the ulcer → produces pain (usually occurs at night → **characteristic** of DU)

d. Relieved by : food and by antacids (patients carry with them biscuits that they consume when pain comes on)

2. Nausea and vomiting

Not prominent features of uncomplicated ulcers.

3. Periodicity.

-pain occurs during periods of activity of the ulcer (variable length but usually last for a few weeks)

-followed by a period of quiescence (lasts for a few months) during which these patients are quite healthy.

4. Clinical picture of complications (may be the 1st presentation)

a. Anaemia with easy fatigue and pallor due to chronic bleeding

b. Melaena and/or haematemesis with shock due to acute bleeding

c. Repeated vomiting due to pyloric stenosis

d. Sudden severe pain of peritonitis when ulcer perforates

e. Pain referred to the back (means penetration of the pancreas)

Signs:

Localized epigastric tenderness may be present

Investigations

1-Endoscopy (**best investigation**)

-Done in all patients suffering from typical ulcer dyspepsia.

- the ulcer is directly visualized

-Advantage → no radiation

2. Barium meal (2nd place after endoscopy).

-Normal duodenal cap appears as a smooth triangle

-The ulcer itself may show as:

a) A small barium spot that remain inside the ulcer crater after the passage of barium.

b) A fixed pouching of the duodenal cap

c) Fixed deformity (**trefoil**) if fibrosis occur

3. To detect *Helicobacter pylori*:

a) Culture from biopsy material (more commonly)

b) Breath test c) serology

Treatment

Medical treatment (The main treatment of uncomplicated cases)

-The aim: reduce acidity to heal the ulcer & to maintain it healed

-Medications:

1- Proton pump inhibitors (PPIs)

- Examples: oral omeprazole (20 mg/day) and lansoprazole.

-Mechanism : markedly reduce gastric acidity in a matter of days.

- IV omeprazole is used emergencies.

Duodenal cap is normally triangular

Normal duodenal cap



Fixed duodenal out pouch



Trefoil deformity



- NSAIDs are prohibited.
-Endoscopy is repeated after 6 weeks of medical treatment to evaluate healing, if ulcer healed →full dose ttt is continued for few more weeks
- The main disadvantage of medical treatment is that once the drugs are stopped → high rate of symptoms recurrence

2- H₂-receptors blockers

- Examples: cimetidine, ranitidine and famotidine.
- Mechanism: reduce gastric acid secretion
- The usual dose of oral ranitidine: is 150 mg twice daily.

3-Eradication of *Helicobacter pylori*:

- Its eradication reduces the recurrence rate
- A triple drug therapy consists of :
 - a) Metronidazole
 - b) tetracycline or amoxicillin
 - c) bismuth compounds
- given for 2 weeks → excellent eradication in 90% of cases.
- Testing for *Helicobacter* repeated 4 weeks after stopping treatment

Surgical treatment

- Is mainly indicated for complicated cases
- Elective surgery is indicated for :
 1. Failure of medical treatment to control symptoms
 2. Relapses after repeated attempts of adequate medical treatment.
 3. A duodenal ulcer presenting by fibrotic pyloric stenosis
 4. Complicated peptic ulcer (perforation, Bleeding or obstruction).

-Aim of surgery:

- 1- Heal the ulcer
- 2- Adequate protection against recurrence.

The principle of surgery:

Vagotomy→ permanent & adequate reduction of gastric acid secretion, which may be:

A-Truncal vagotomy and drainage

- The trunks of the vagus nerve are exposed & cut → stomach denervation →abolish gastric acid secretion immediately by **80%** decreasing to **50%** or less by time (not compromise surgery results)
- Recurrence rate about 5-10%.
- Denervation of the antrum leads to gastric stasis so drainage procedure is needed such as either:

1- Gastrojejunostomy

- Creating a stoma between a dependent part of the stomach and the proximal jejunum.

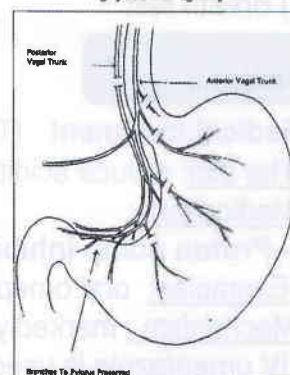
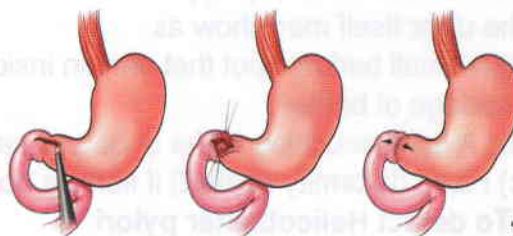
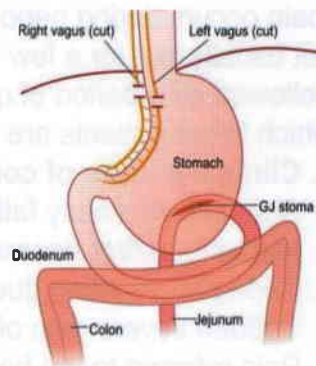
2- Pyloroplasty.

- destroy the pyloric sphincter by cutting it longitudinally then sutured transversely

B-Highly selective vagotomy

- denervate only the body and fundus of stomach (the acid secreting parts) leaving the pylorus and its antrum fully innervated → preserving gastric emptying (no need for drainage procedure)
- ligate all the branches of the left gastric artery to the stomach (because the secretory fibres run in close association with them), stopping short of the branches of the antrum (**The Crow's foot**)

Vagus is responsible for nervous phase of gastric acid secretion



Chronic Gastric Ulcer

Types

3 types

• Type I ulcers

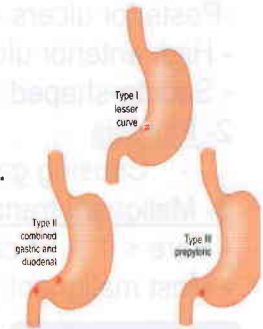
- Occur in the body of the stomach on the lesser curvature (60%).
- Preceded by gastritis that causes damage to the gastric mucosal barrier.
- Not caused by high acid production (patients have lower than normal acid secretion)

• Type II ulcers

- occur in association with a duodenal ulcer (20%).
- The duodenal ulcer develops 1st → induce duodenal deformity and gastric stasis → gastric ulcer

• Type III ulcers

- occur in the antrum or pyloric canal (20%).
- Type II and III ulcers are associated with gastric hypersecretion and have the same etiology as duodenal ulcers



-Incidence:

1. Less prevalent than duodenal ulcer.
2. Males > females (by a smaller margin than in DU)
3. GU occurs at older age than DU (exceptions occur)

- An ulcer situated away from the lesser curvature is highly suspicious of malignancy

- Diagnosis of a gastric ulcer is not problematic but the problem is to be certain whether that ulcer is malignant or not

Etiology

- Not exactly known, causes of gastritis precede ulceration are:

1. Reflux of bile into the stomach

- cause damage to the gastric mucosal barrier → H⁺ ions back diffuse → mucosal damage.

2. Antral stasis

- due to defective gastric emptying → hypergastrinaemia

3. Helicobacter pylori

- produce gastritis but its exact etiological role not known

4. NSAID

- Increase risk of GU especially if with Helicobacter infection.

5. Irritant substances

- As smoking (inhibits ulcer healing), aspirin and alcohol

Pathology

Gross appearance:

- Size: larger than DU

- Site:

- 1- at the junction of the body and antrum on the lesser curvature
- 2- Some ulcers are saddle-shaped overriding the lesser curvature.

- Edge and floor:

Sloping edges & floor covered by granulation tissue.

- Base: -There is marked induration around it.

- Usually lies in the muscle layer but may lie in any tissue the ulcer has penetrated as the pancreas.

Complications

Acute

1- Perforation

- If perforation occurs posteriorly → it occurs in the lesser sac.

2- Bleeding



Chronic

1-Penetration

- Posterior ulcers → penetrate the pancreas
- High anterior ulcers → penetrate the liver
- Saddle-shaped ulcers → penetrate both the pancreas and the liver.

2- Fibrosis

- Causing gastric outlet obstruction or **Hour-glass stomach**.

3- Malignant transformation

- Rare < 1 % of cases.
- Most malignant gastric ulcers are malignant from the start



Clinical picture

Type of patient:

Elderly that appears thin and underweight.

Symptoms:

1. Pain.

- Character: boring
- Location: deeply seated in the epigastrium
- Time: starts shortly or immediately after food intake so the patients are afraid to eat → lose weight.
- Periodicity: - less well marked than in DU
- Its loss may indicate malignancy.
- Relieved by: antacids, fasting and vomiting

2. Nausea and vomiting

Vomiting relieves pain and may be self induced.

3. Clinical picture of complication

- a) Pain referred to the back → indicating penetration.
- b) Bleeding and perforation
- c) Fibrous contractures → **hour-glass stomach**.

Signs

Localized epigastric tenderness

- Gastric ulcer pain does not occur when the stomach is empty → no night pain as in DU.
- If antacids fail to relieve the pain → possibility of malignancy or penetration



Investigations

1. Endoscopy (The **main** investigation)

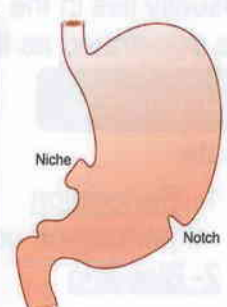
- Directly visualized ulcer and its characteristics noted
- Multiple biopsies (at least 4) obtained to exclude malignancy.

2. Barium meal (2nd place after endoscopy)

- An **ulcer niche** → an outpouching on the lesser curvature or a crater that remains filled with barium after its passage from the stomach
- **Ulcer notch** → area of spastic muscle on the greater curvature opposite the ulcer niche

3. Tests for *Helicobacter pylori*.

- a) Culture from biopsy material (more commonly)
- b) Breath test
- c) Serology



I-Medical treatment:

(Exactly similar to DU but because of risk of being malignant → must exclude malignancy before giving medications)

1- Proton pump inhibitors (PPIs)

- Examples: oral omeprazole (20 mg/day) and lansoprazole.
- Mechanism: markedly reduce gastric acidity in a matter of days.
- IV omeprazole is used emergencies.

2- H₂-receptors blockers

- Examples: cimetidine, ranitidine and famotidine.
- Mechanism: reduce gastric acid secretion
- The usual dose of oral ranitidine: is 150 mg twice daily.

3- Eradication of Helicobacter pylori.

- Its eradication reduces the recurrence rate
- A triple drug therapy consists of :
 - d) Metronidazole
 - e) tetracycline or amoxicillin
 - f) bismuth compounds
- given for 2 weeks → excellent eradication in 90% of cases.
- Testing for Helicobacter repeated 4 weeks after stopping ttt

II-Surgical treatment:

Indications for surgery:

1. Failure of healing to start at 6 weeks or to complete at 6 months after medical treatment.
2. Uncertainty about the absence of malignancy.
3. Complications of gastric ulcer that require surgery.

Aim of surgery for benign gastric ulcers:

Remove the ulcer or the entire ulcer bearing area to ensure proper gastric emptying.

Operation:

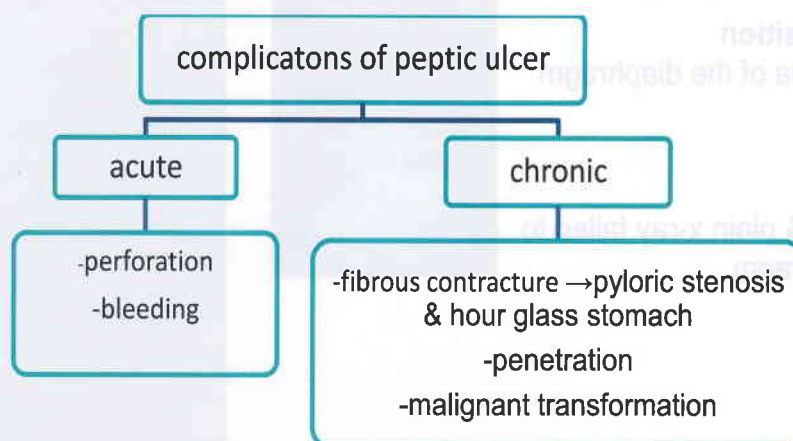
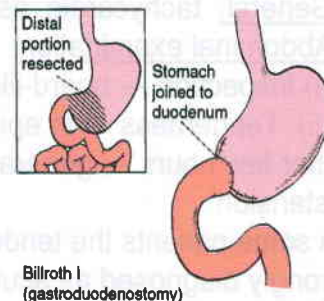
Partial gastrectomy (the standard procedure)

- It removes the ulcer together with the entire ulcer-bearing area.
- ended by gastroduodenal anastomosis → **Billroth I**

- Repeat endoscopy after 6 weeks regardless of symptomatic improvement, If:

- a) The ulcer shows no attempt at healing → considered malignant and surgery performed.
- b) Partial healing is seen → continue medical treatment for another 2 months (never > 6 months) & repeat endoscopy → if complete healing not achieved → surgical treatment

- Some patients with gastric cancer may improve on medical treatment



Perforation

Pathology

- Depending on the degree of aggressiveness of the ulcer, perforation will be:

- a) Rapid → generalized peritonitis
 - b) Slow → small and are rapidly sealed.
- Occur more in anteriorly situated ulcers.

- Stages:

a) Chemical peritonitis

At time of perforation → duodenal or gastric contents (sterile) burst into the peritoneal cavity → inflammatory response.

b) Bacterial peritonitis

- After few hours → swallowed bacteria (with saliva) and bacteria from the gut → gain access to the peritoneal cavity (through the Perforation) → turn chemical peritonitis into bacterial peritonitis.

Clinical picture

Symptoms

- Sudden severe pain (at the time of perforation) starting in epigastrium and later becomes generalized.
- The patient often collapses especially with a large perforation.

Signs

- General: tachycardia, rising pulse rate and pyrexia

- Abdominal examination:

- i) Inspection → board-like rigidity
- ii) Tenderness → in epigastric region.

.After few hours → generalized abdominal tenderness, rigidity and distension.

.In some patients the tenderness is marked in the right iliac fossa → wrongly diagnosed as acute appendicitis

- iii) Auscultation → absent bowel sounds.

Investigations

(Urgent)

1. Chest x-ray in erect position

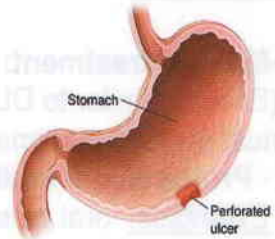
Reveals air under the cupula of the diaphragm

2. Abdominal ultrasound

Reveal peritoneal fluid

3. Gastrograffin meal

If perforation is suspected & plain x-ray failed to reveal gas under the diaphragm



- The inflammatory exudate at 1st collects in the supracolic compartment of the peritoneal cavity → soon spills to the infracolic compartment through RT paracolic gutter (left closed by the phreno-colic ligament) & may remain localized to RT iliac fossa → wrongly diagnosed as acute appendicitis

- Small perforations may be sealed rapidly without much soiling of the peritoneal cavity

-Differential diagnosis of perforation:

A- From causes of acute upper abdominal pain.

- 1- Acute cholecystitis.
- 2- Acute pancreatitis.
- 3- Mesenteric vascular occlusion
- 4- Leaking aortic aneurysm.
- 5- Basal MI

B- From acute appendicitis if it presents by RT iliac fossa pain.

Treatment

(Urgent operation after preparation)

Preoperative preparation includes:

1. A Ryle → continuous aspiration
2. IV fluids (are guided by urine output, electrolyte and pH)
3. IV antibiotics
4. IV proton pump inhibitors
5. Careful attention to respiration (is often compromised)

The operation: (the simplest procedure is the best)

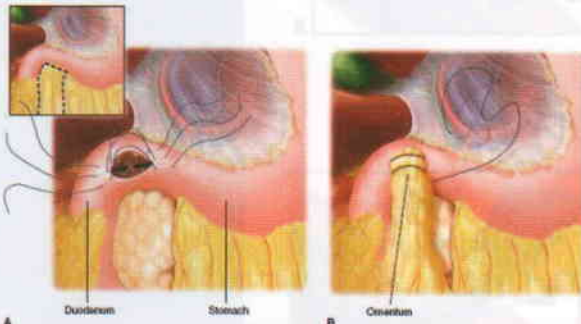
- 1- Thorough peritoneal toilet
- 2- Closure of the perforation with an omental patch

(Graham's method)

- 3- The abdomen is closed with drainage.

Post operative

Medical treatment for the ulcer



Bleeding

Pathogenesis

Haematemesis and melaena

- Occurs due to → ulcer eroding a large vessel at its base.
- a small erosion → bleeds a little then sealed, may be repeated
- a large erosion in the vessel wall → massive bleeding.

Symptoms:

1- Haematemesis

Vomiting a small dark brown-coffee ground-blood

2- Melaena → passage of loose black tarry offensive stools

3- Bleeding per rectum

Fresh red blood may be passed with stools if bleeding is severe

Signs:

In severe cases → Hypovolaemic shock (pale, cold, a rapid pulse & low blood Pressure)

Investigations

CBC

Hb & haematocrit useful only after a few hours → hemodilution

- Aim of preoperative preparation is to correct hypovolaemia & electrolyte imbalance
- If treatment is delayed → hypovolaemic or septic shock will occur.
- In the case of a perforated gastric ulcer a biopsy must be obtained from it

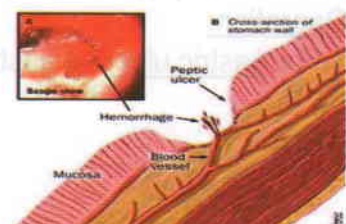
Subacute perforation.

- This is a small perforation → only a minimal amount of contents enter the Peritoneal cavity and is rapidly sealed.
- It gives the same early signs as acute perforation but much reduced .

- If diagnosed correctly → treatment is conservative

Chronic perforation

- means penetration into an adjacent structure as the pancreas.
- It is suspected when the pain becomes more or less persistent & referred to the back



A-Conservative treatment: (considered whenever possible)

1-Stabilize general condition

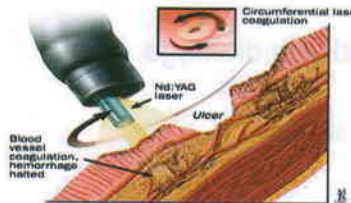
- Admission to hospital (better to an ICU)
- High-flow oxygen by mask.
- Correction of hypovolaemia by IV crystalloids and blood
- Monitoring of urine output, pulse, blood pressure & haematocrit
- Nasogastric tube.

2- IV PPIs.

3- Urgent endoscopy (done once general condition is stabilized)

-Findings may be seen:

- An ulcer with oozing artery
- A visible vessel in the ulcer base
- Fresh blood clot covering the ulcer.

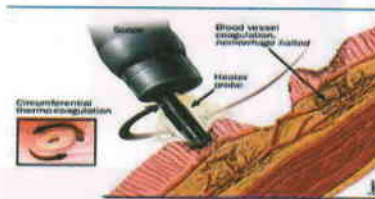


Technique

Fine instruments are introduced through the channel of the Gastroscope → reach the ulcer → arrest bleeding

Methods

- Laser coagulation
- thermal coagulation
- Diathermy coagulation
- Injection of the ulcer base by adrenaline or alcohol.



B-Surgical treatment

Preoperative preparation

- The patient should have more than one IV line open and running
- A nasogastric tube is passed → stomach is washed & emptied.

Indications:

Absolute indications:

- No improvement under adequate medical treatment
- The initial bleed was severe → 2000 ml or more.
- Continuous bleeding that needs transfusing large volumes (1000 ml) of blood per day to maintain stability.
- If bleeding recurs after endoscopic homeostasis.

Relative indications:

1. Old atherosclerotic patients

Because 1) they can't withstand the ill effects of shock

2) Their arteries can't retract enough to stop hemorrhage.

2- Long history of ulcer disease

Do not respond well to conservative treatment.

2. Those with associated serious disease.

The risk of operation is less than the risk of shock.

Operation:

- For gastric ulcer → gastrectomy (the standard operation)

-Bleeding from a PU always occurs but only on a microscopic level & Considered part of the disease process

-Small bleeds cause no alteration in general condition but they require hospitalization & receive exactly the same diagnostic and treatment policies as large bleeds

- Most of cases stop bleeding

spontaneously due to clot formation in the base of the ulcer → obstructing the erosion in the artery

- Cases that usually respond favorably to conservative treatment:

1. Young patients < 45 years.

2. Minor bleeds.

3. Short history of dyspepsia.

4. Recent history of ulcerogenic drug intake (drug withdrawal is all what is required)

-The decision to operate should be taken early (within 48-72 hours) as the results of late surgery are poor with a high mortality

2- For duodenal ulcer:

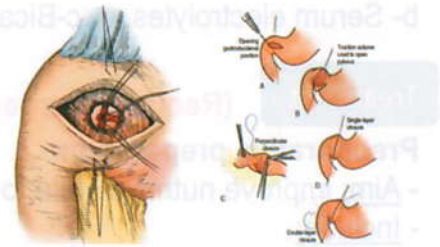
a- **Pyloroplasty**

i) The pylorus is opened **longitudinally** to expose the bleeding ulcer.

ii) Homeostasis is achieved by under- running sutures

iii) The pylorus is then closed **transversely**

b- **Truncal vagotomy** (+ / -) → to reduce acid secretion.



Pyloric Stenosis

Pathogenesis

- Fibrosis around DU or juxta-pyloric ulcer → gastric outlet obstruction.
- Usually occurs over years of ulcer dyspepsia (may occur silently)

Clinical picture

Symptoms :

1. Vomiting.

- Copious amounts of foul dark fluid characteristically in the evening. (Recognized undigested food eaten the previous day)
- Projectile and does not contain bile.

2. Pain

- Initial periodic pain is lost and is replaced by epigastric discomfort.

Signs :

i) General

- Under weight, ill nourished and may be dehydrated.
- long-standing cases → electrolyte disturbances as hyponatraemia, hypokalaemia, alkalosis and hypocalcaemia (→ tetany)

ii) Abdominal examination

- 1- May show the contour of the enlarged stomach
- 2- Visible peristalsis passing from left to right.
- 3- A succussion splash can often be heard.

Differential diagnosis

Carcinoma of the pyloric antrum

Investigations

1- **Radiological** → **Barium meal** will show:

- Dilated stomach often reaching the pelvis
- Soup dish appearance (fluid level + rugae)
- Delayed gastric emptying & food residue

2. **Instrumental** → **Endoscopy** will show

- A stenosed inactive pyloric ring and will not pass the tip of the endoscope.

- The main value is to detect the presence of a tumour at the pyloric end of the stomach and to obtain biopsies from it if found.



- The patients are usually capable of eating breakfast then have a small lunch but unable to eat in the evening because of a sense of discomfort & fullness which is due to a full stomach

- Barium meal will often miss a small Pyloric carcinoma so Gastroscopy is essential for diagnosis

- Some cases of pyloric stenosis occur due to muscle spasm & a marked Inflammatory reaction around an active duodenal ulcer → responds well to medical treatment as for uncomplicated ulcers

3. Laboratory

- a- CBC → Haemoglobin & haematocrit
- b- Serum electrolytes c- Bicarbonate

Treatment

(Requires surgery but is not an emergency)

Preoperative preparation:

- Aim: improve nutritive state to reduce morbidity and mortality
- Include:
 1. A high protein fluid diet given I.V
 2. Correction of fluid, electrolytes (Na, K, Cl and Ca) and acid-base balance (alkalosis) by proper I.V solutions.
 3. Correct anaemia by transfusion of packed cells
 4. Chest physiotherapy and antibiotics
- Often have pneumonitis due to inhalation
- High risk of postoperative chest infection due to debilitated state.
- 5. Gastric lavage through a nasogastric tube.

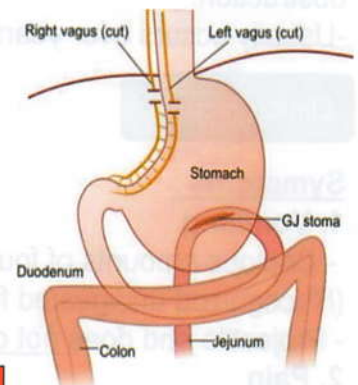
Operation:

1- the standard treatment

Truncal vagotomy and gastrojejunostomy

2- In the elderly or unfit patients do either :

- i) gastrojejunostomy alone
- ii) non operative approach → endoscopic balloon dilatation



Hour glass stomach

- Etiology: a cicatrized saddle shaped ulcer on lesser curvature.
- Symptoms: of gastric obstruction but there will be early satiety and loss of weight.
- Usually occurs in the elderly → should be differentiated from carcinoma of the stomach by endoscopy and a barium meal
- Treatment: by partial gastrectomy (remove the distal pouch)



Complications of gastric operations

A-Early complications:

1. **Bleeding**

- Source: from the anastomotic line
- Diagnosed by: fresh blood being aspirated through the Ryle's tube.
- Treatment: conservative , if failed → re-operation.

2. **Stomal obstruction**

Cause	treatment
a) edema at the stoma(Most cases)	aspiration and IV therapy
b) sometimes after vagotomy (stoma is patent but the stomach is atonic and does not empty	
c) technical error	Surgery to correct them
d) obstruction of gastroduodenal anastomosis by hypertrophied antral mucosa membrane	

3. Duodenal blow-out (follows a Billroth II anastomosis)

- Timing: around the 4th day (may occur later)

-Causes:

- a. Sutures taken in an inflamed or a densely scarred duodenum

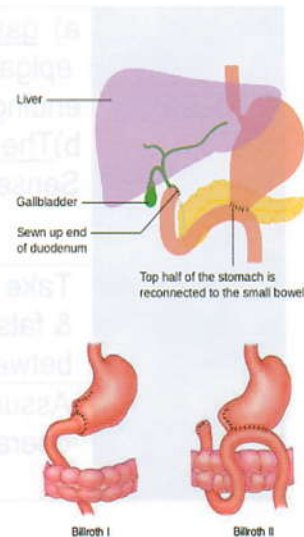
b. Technical error:

- i) Obstruction of the afferent loop
- ii) Excessive turning of duodenum
- iii) Too aggressive dissection → Ischaemia.

-If no drainage was provided → acute severe upper abdominal and lower chest pain with anxiety & tachycardia → generalized peritonitis

- If drainage was provided with I.V fluids

- i) Without distal obstruction → all cases are cured
- ii) With distal obstruction → reoperation to relieve afferent loop obstruction



4. Post-operative pancreatitis

B-Late complications:

1. Recurrent ulceration:

-Causes:

- a) Inadequate gastrectomy (leaving part of the gastric antrum with the duodenum) → persistent hypergastrinaemia.
- b) Missing a trunk during vagotomy
- c) Recurrence of the original ulcer

- Site: may be Stomal (on the anastomotic line or on one side of it whether gastric or jejunal)

-Clinical diagnosis:

- a) Recurrence of typical ulcer symptoms
- b) Pain may be referred to bizarre areas as hypochondrium or loin.

- Diagnosis: Endoscopy is essential

- Treatment:

- i) **Medical** → Proton pump inhibitors
 - Will control almost all patients
 - Recurrence is common after drug cessation → reoperation is usually needed.

- ii) Surgical (depends on previous operation done)

- a) Following vagotomy → if there is missed trunk → divide it
→ No missing trunk → do antrectomy

- b) Following gastrectomy → do vagotomy.

2. **Dumping:**

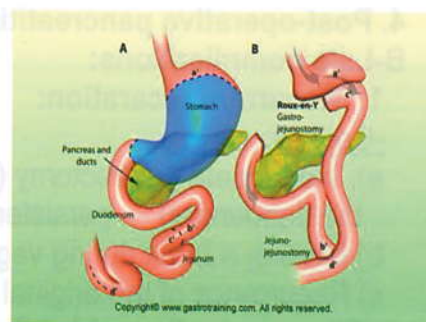
- It has 2 components: vasomotor and gastrointestinal symptoms following meals.

	Early dumping	Late dumping
Timing	within the first 1/2 hour after meal	about 2-3 hours after meal
Cause	Rapid gastric emptying → delivery of hyperosmolar solution to the proximal small gut → shift of fluid from the circulatory plasma to the lumen of the bowel → vasomotor effects	Rapid delivery of large amounts of carbohydrates to the bowel → overshoots of insulin secretion → hypoglycaemia.

Clinical picture	a) <u>gastrointestinal</u> : epigastric fullness & pain with nausea ending in explosive diarrhoea. b) <u>The vasomotor</u> : Sense of weakness, flushing & palpitation.	<u>Symptoms of hypoglycemia</u> 1. sweating 2. palpitation 3. tremor 4. confusion
Prevention	Take small frequent meals rich in proteins & fats and less in CHO with liquids only between meals.	frequent small meals of a low carbohydrate diet
Treatment	-Assume trendelenburg position -operation rarely needed	carbohydrate ingestion

3. Biliary gastritis (alkaline reflux gastritis):

- Definition: Reflux of bile through a stoma in the stomach → diffuse gastritis.
- Investigations: endoscopy is mandatory
- Treatment: only by surgery → Fashioning of a Roux-en-Y anastomosis



4. Postvagotomy diarrhea:

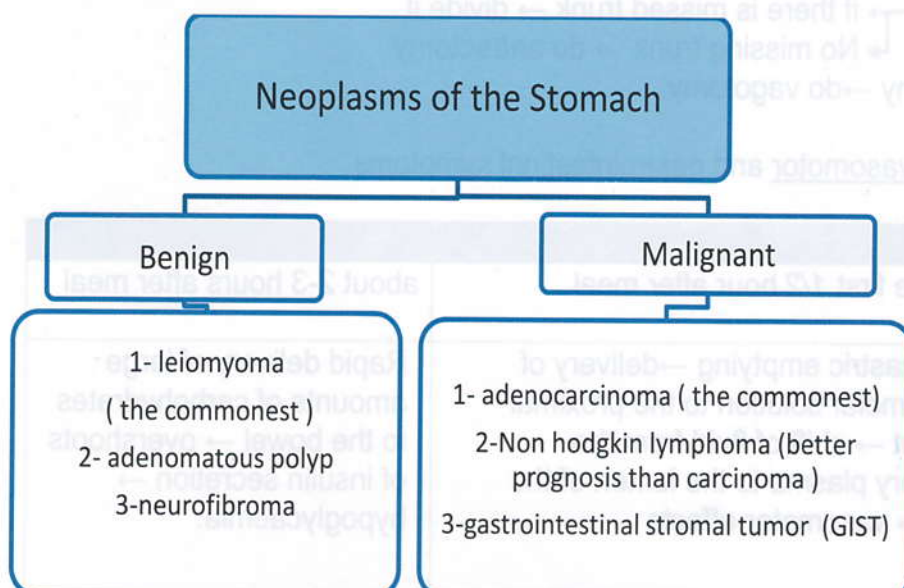
- A tendency to loose stools follows truncal vagotomy in most cases.
- Incidence: is not common in Egypt.
- Etiology: is obscure but may be due to dysfunction of the ileocaecal valve → bile acids being delivered too rapid to the colon
- Treatment: gets better by time

5. Postgastrectomy nutritional disturbances:

- They are: a) Weight loss b) steatorrhoea
- c) Anaemia (iron deficiency or megaloblastic)
- d) Vitamin B deficiency e) Calcium deficiency
- f) Early satiety g) Increased risk of pulmonary TB.

6. Increased risk of stomach cancer following gastrectomy

- Biliary gastritis mimics recurrent ulceration but with the symptom of bilious vomiting.
- Rarely in about 3% of cases, the diarrhoea is watery and explosive



All types of benign neoplasms expected to arise from mucous membrane, muscle, & interstitial tissue occur in the stomach.

leiomyoma

- The commonest benign tumor
- Symptoms: vague
- Investigations: barium meal → smooth filling defect
- Complications:
 - a) It can bleed or may perforate the stomach.
 - b) It occasionally gives rise to an epigastric mass.
 - c) Malignant transformation can occur
- nowadays considered as a type of GIST.



Figure 2. Barium meal film showing a filling defect in the fundus of the stomach, a common position for leiomyoma.

Carcinoma of the Stomach

Etiology

1. **Chronic irritation** by:
 - a) Dietary as spices, increased salt intake, tobacco, alcohol, N-nitrosamine compounds
 - b) Following partial or subtotal gastrectomy (**≈20 years**) biliary reflux gastritis → remaining gastric stump carcinoma.
2. **Chronic lesions**
 - a) Chronic H. pylori infection (increases risk about **3 times**)
 - b) Factors that cause achlorhydria such as viral infections
 - c) Benign gastric neoplasms as gastric polyps
 - d) Benign gastric ulcers (very rarely)
- 3- **A genetic predisposition.**
 - a) Family history
 - b) Persons with blood group A
 - c) Pernicious anaemia

Pathology

Site:

- **50%** near the cardia
- **35%** occur in pyloric antrum
- **10%** diffuse infiltration of the whole stomach (**linitis plastica**).

Macroscopic types: 2 types

A. Early gastric cancer

- Only the mucosa or submucosa is infiltrated.
- Only diagnosed if screening programs done by endoscopy
- May be protruding, superficial, or excavating (penetrating).

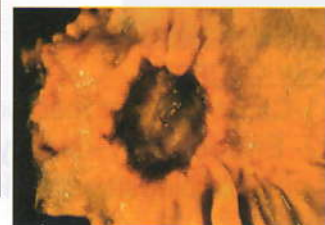
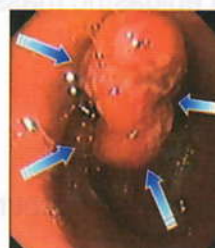
B. Advanced gastric cancer

(The usually diagnosed type in practice)

- Forms:

1. A fungating cauliflower-like mass
2. An ulcer with raised indurated edges
- c. Colloid carcinoma.

All layers of the stomach are infiltrated by areolar tissue containing transparent gelatinous substance.



-Incidence:

Has been falling steadily throughout the world except in Japan (is of almost epidemic proportions; 100/100,000 of the population)
-In general any factor causes gastritis can cause carcinoma.
-presence of nitrates in food leads to the production of N-nitrosamines by the action of bacteria in the achlorhydric stomach
- Whatever the actual factor, carcinoma will occur if there is gastritis & a genetic predisposition

d. The diffusely infiltrating variety. "**Linitis plastica**"

- The wall of the stomach is greatly thickened and indurated while the lumen is greatly reduced.
- May occur only in the antrum or the stomach diffusely.
- Mucous membrane is intact & may be missed by endoscopy.

Microscopically

1- Adenocarcinoma (**commonest**)

- has various grades of differentiation → the less the differentiation the worse the prognosis.

2- A colloid carcinoma → bad prognosis

3- Linitis plastica: difficult to find malignant cells under the microscope → bad prognosis



Spread

1. Direct

- Infiltration of the wall of the stomach in depth, circumference & length
- After that will infiltrate neighbouring structure as the liver, spleen, pancreas or colon.

2. Lymphatic

- By both permeation and by embolization.
- To perigastric L.Ns (1st) then to more centrally placed groups.

3. Blood

- Mainly to the liver (very rarely beyond it to bones)

4. Transcoelomic

- Krukenber's tumour: ovarian secondaries in younger females when the ovaries are not yet atrophic
- Blumer's shelf: Metastases to the Douglas Pouch

Clinical picture

1- Dysphagia

With cancer near the cardia

2- Gastric outlet obstruction

With a tumour near the pylorus

3- Symptom of advanced disease

- a) vague discomfort after meals
- b) anorexia
- c) rapid weight loss
- d) vomiting

4- Uncommon presentations

- a) Haematemesis
- b) Melaena
- c) An epigastric mass.

Investigations

For diagnosis

1- **CBC**: micro or macrocytic anaemia.

2- **Endoscopy**: -directly view the tumour from which
-multiple biopsies should be taken.

-The length spread is important because it occurs for some length beyond the edge of the tumour → important in radical operation

- knowing the lymphatic drainage of the stomach is important in planning radical surgery

- Early symptoms of gastric cancer are vague and are usually neglected by patients & not recognized by the physicians.

- Approximately 10% of patients present with one or more sign of metastatic disease as metastases in the liver, enlarged supraclavicular lymph node, ascites or an umbilical nodule

- **Anemia is the earliest sign in gastric carcinoma**

- Endoscopy can detect tumours at an earlier stage than can radiology

3. Barium meal.

- Its overall accuracy is about 75%.
- should always include views taken in the Trendelenburg's position
→ to detect growths situated in the fundus and cardia.
- It shows:

- a) In cauliflowerlike mass → persistent irregular filling defect
- b) In malignant ulcer → ulcer niche outside the ulcer bearing area.
- c) In linitis plastica → marked narrowing of the lumen of the stomach but the flow of barium is not interrupted

4- Diagnostic laparoscopy.

For staging

1. An abdominal sonogram

- For the detection of liver secondaries.
- Assess lymph node involvement (less important)

2- CT scan

- Detect L.N.S involvement (more accurate)
- Helpful in pre-operative staging of the disease.

3- Endoscopic ultrasound

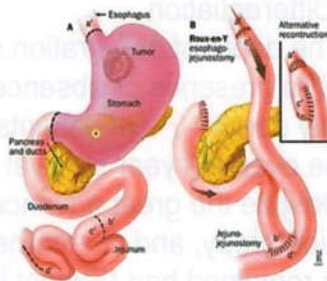
- Detect the depth of invasion of the gastric wall

4- PET -CT scan

- To detect metastases

For follow up

Tumor markers → **CEA**

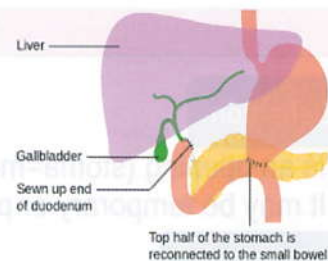


Treatment

A- Early cases.

-The aim: is cure the patient

- achieved by radical operation, this includes removal of :
 - 1) The tumour with an adequate safety margin of at least 5 cm above it and 1.5 cm of the duodenum below it
 - 2) Both omenta (greater & lesser)
 - 3) All the draining lymph nodes with ligation and division of the vessels they are located around as: the left gastric, the right gastric and the two epiploics.
- The extent of resection depends on site of the neoplasm:



Site	Operation	anastomosis
Lower 1/3	a lower radical partial gastrectomy	the remaining upper stomach to the jejunum. (Billroth II or Polya gastrectomy)
Middle 1/3	total radical gastrectomy	oesophagus to a Roux-en-Y loop of jejunum
Upper 1/3	oesophagogastrectomy through a thoracoabdominal incision to guarantee an upper safety margin	oesophagus is anastomosed to a Roux-en-Y loop of jejunum

B-Patients with incurable or irresectable lesions

May benefit from various palliative procedures as:

I. For resectable tumors

Palliative gastrectomy (best palliation)

II-For irresectable tumors

1- In the pyloric region → **anterior gastrojejunostomy**

2-In the upper stomach → **oesophagojejunostomy**

III-For inoperable, non resectable tumors, incompletely resected or recurrent cases → Radiotherapy and chemotherapy

(Unfortunately offer too little for these miserable patients)

Prognosis

-Depends on many factors the most important are:

1- The histological type

2- Differentiation

3-The depth of penetration of the wall

4- The presence or absence of lymph node involvement.

-Only about **40%** of patients are found to be operable at exploration.

-The overall 5 year survival is **10-20%**.

- Despite the great advances that have occurred in surgery, radiotherapy, and chemotherapy, the prognosis of gastric cancer has remained bad (Except in Japan due to screening programs → early detection)

Gastrostomy

Definition

- Is an opening (stoma=mouth) of the stomach on the skin.
- It may be temporary or permanent

Indications

1-To decompress the bowel as an alternative to postoperative nasogastric intubation

- Advantages:

a) More comfortable

b) Does not interfere with deep respiration & coughing which are encouraged to avoid pulmonary complications.

c) Of advantage in the elderly with chronic pulmonary disease.

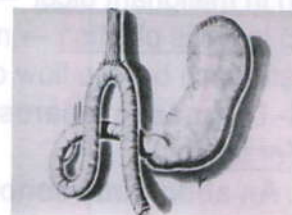
2. To feed patients who cannot ingest food orally for weeks or months as in operations on the mouth and pharynx

Technique

1-**Stamm's gastrostomy** (The traditional method)

a. A small puncture is made in the middle of the anterior surface of the stomach.

b. A large **Malecot** (Mushroom head) or de Pezzar tube is inserted 1 or 2 inches into the lumen



Gastrointestinal Stromal tumours (GIST), formerly leiomyoma

-Site: Common in the stomach & S.I.

-Source: It arises from **Cajal cells** (lie within the stomach wall)

- Symptoms, may present with:

- a) Abdominal mass
- b) Haematemesis
- c) Dyspepsia.

- May reach a large size.

- May have a pseudocapsule

- Treatment:

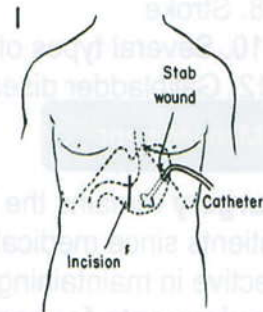
1-by local gastric resection

(recurrence is common)

2- **Gleevac** → a tumour specific monoclonal antibody against some tyrosine kinases of the tumour (shown excellent clinical results)



- c. Two concentric purse string silk sutures are used to invert the stomach wall → a snug seal and a serosal lining to the tube tract.
- d. The tube is brought out through a tightly fitting stab wound in the left upper quadrant.
- e. The stomach is firmly attached to the abdominal wall by sutures around the exit site of the tube to prevent early retraction and leakage into the peritoneum.
- f. When it is no more needed, the tube is simply pulled out → track closes spontaneously because it is lined by serosa.

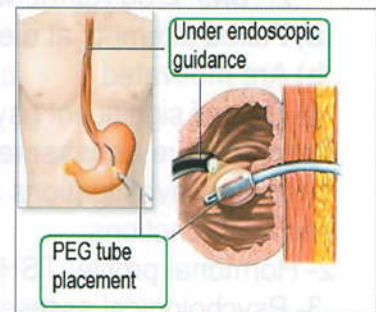


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2. Percutaneous endoscopic-assisted gastrostomy (PEG)

Is increasing popularity

- a. A gastroscope is inserted into the stomach and is used to inflate it.
- b. The gastroscope light is made to shine through the anterior stomach wall and anterior abdominal wall
- c. Under local anaesthesia a trocar and cannula puncture the anterior abdominal wall and enter the stomach
- d. A self-retaining tube is introduced through the cannula into the stomach and its balloon is inflated



Bariatric Surgery

Etiology

involve genetic, environmental, metabolic and psychological factors.

Grades of obesity

- measured by calculating the body mass index (BMI)

$$\text{BMI} = \frac{\text{weight (in Kg)}}{(\text{height in m})^2}$$

	Obesity class	BMI (kg/m ²)
Underweight		<18.5
Normal		18.5-24.9
Overweight		25.0-29.9
Obesity	I	30.0-34.9
Severe obesity	II	35.0-39.9
Morbid obesity	III	40.0-49.9
Super-morbid obesity	IV	> 50

Complication

Comorbid diseases which affect nearly every organ system as:

1. Type 2 diabetes
2. Cardiovascular disease
3. Hypertension
4. Hyperlipidemia
5. Hypo-ventilation syndrome
7. Sleep apnea
6. Asthma

- Obesity is a pan-endemic health problem worldwide

- Obesity shortens life expectancy with increasing body mass index (BMI) → proportionally shorter lifespan.

- Morbid obesity is projected to overtake smoking as the leading cause of death in the near future

- Non-operative management with diet, exercise, behaviour modification & medications rarely achieves adequate weight loss.

- medical treatment once stopped → weight is regained

8. Stroke
10. Several types of cancers
12. Gallbladder disease
9. Arthritis
11. Urinary incontinence
13. Depression

Management

- **Surgery** remains the best option for many morbidly obese Patients since medical therapies have not been shown to be effective in maintaining long-term weight loss in a morbidly obese

Requirements for bariatric surgery

- 1- BMI >35 Kg/m²
- 2- BMI ≥ 30 Kg/m² with co-morbidities provided that :
 - a) Failed attempts at diet and exercise
 - b) Are motivated
 - c) Are well informed
 - d) Free of significant psychological disease.

Preoperative assessment and investigations

- 1-Routine investigations as CBC, blood sugar, urea & electrolytes and liver functions
- 2- Hormonal profile: TSH, T4 and plasma cortisol.
- 3- Psychological assessment.

Bariatric Surgery Procedures

Bariatric surgical procedures cause weight loss by

1. Restricting the amount of food the stomach can hold
2. Causing malabsorption of nutrients
3. Combination of both gastric restriction and malabsorption.

Laparoscopic Roux-en-Y gastric bypass (LRYGB)

The gold standard surgery and the most commonly performed

Advantages

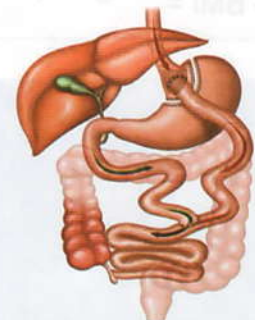
- 1- Produces significant long-term weight loss (60 to 80 %)
- 2- Restricts the amount of food that can be consumed
- 3- May lead to conditions that increase energy expenditure
- 4- Typical maintenance of >50% excess weight loss
- 5- less absorption of calories nutrients as it bypass of a large segment of small bowel
- 6- rerouting of the food stream → changes in hormones (ghrelin and Leptin) → promote satiety & suppress hunger

Disadvantages

- 1- Is technically a more complex operation than the AGB or LSG and potentially could result in greater complication rates.
- 2- Can lead to long-term vitamin/mineral deficiencies particularly deficits in vitamin B 12, iron, calcium and folate
- 3- Requires adherence to dietary recommendations, lifelong vitamin /mineral supplementation, and follow-up compliance.

The most commonly performed procedures are:

1. Laparoscopic Roux-en-Y Gastric Bypass (LRYGB).
2. Laparoscopic Mini Gastric Bypass (LMGB),
3. Laparoscopic Sleeve Gastrectomy (LSG).
4. Laparoscopic Adjustable Gastric Band (LAGB).



Laparoscopic Mini Gastric Bypass (LMGB)

A small gastric pouch (50ml) is fashioned along the lesser curvature of the stomach and is anastomosed to a loop of the small bowel 2 meters from the duodenojejunal flexure

Laparoscopic Sleeve Gastrectomy (LSG)

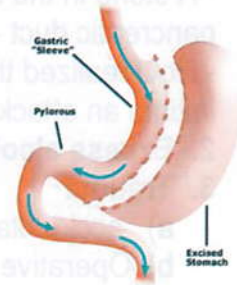
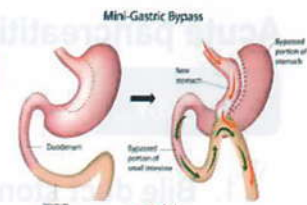
Approximately 80 percent of the stomach removed & the remaining stomach is a tubular pouch

Advantages

1. Restricts the amount of food the stomach can hold
2. Induces rapid and significant weight loss that comparative studies find similar to that of the Roux-en-Y gastric bypass.
3. Requires no foreign objects (AGB), and no bypass or re-routing of the food stream (RYGB)
4. Involves a relatively short hospital stay
5. Causes favorable changes in gut hormones that suppress hunger, reduce appetite and improve satiety

Disadvantages

1. Is a non-reversible procedure
2. Has the potential for long-term vitamin deficiencies



Laparoscopic Adjustable Gastric Band (LAGB)

-An inflatable band that is placed around the upper portion of the stomach → small gastric pouch above the band and the rest of the stomach below the band

-The size of the stomach opening can be adjusted by filling the band with sterile saline which is injected through a port placed under the skin.

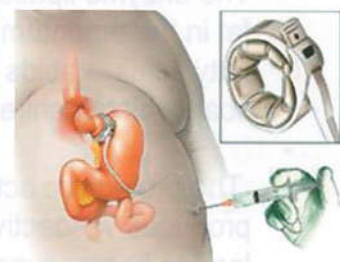
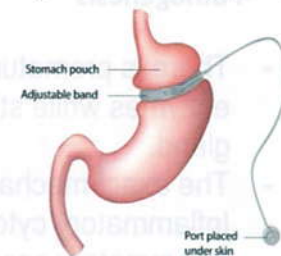
Advantages

1. Reduces the amount of food the stomach can hold
2. Induces excess weight loss of approximately 40 - 50 %
3. Involves no division of the stomach or rerouting of the intestines
4. Requires a shorter hospital stay (< 24 hours)
5. It is reversible and adjustable
6. Lowest rate of early postoperative complications & mortality
7. Has the lowest risk for vitamin/mineral deficiencies

Disadvantages

1. Slower & less early weight loss than other surgical procedures
2. Greater percentage of patients failing to lose at least 50 % excess body weight compared to the other surgeries commonly performed
3. Requires a foreign device to remain in the body
4. Possible band slippage or band erosion (small percent)
5. Mechanical problems with the band, tube or port (small %)
6. Can result in dilation of the esophagus if the patient overeats
7. Requires strict adherence to the postoperative diet and to postoperative follow up visits.
8. Highest rate of re-operation

Adjustable Gastric Band (Lap Band)



Pancreatitis

Acute pancreatitis (Mortality is about 10%)

Etiology

1. **Bile duct stones (50%).**
 - A stone lodged in the extreme lower part of the common bile duct is likely to obstruct the pancreatic duct as well.
 - A stone in the ampulla of Vater → regurgitation of bile into the pancreatic duct → activation of pancreatic enzymes .
 - Now realized that passage of a stone through the ampulla may initiate an attack
2. **Excess alcohol intake (35%).**
3. **Trauma**
 - a) accidental
 - b) Operative
 - c) ERCP → the third most common cause of pancreatitis
4. **Idiopathic (in 20 % of cases)**
5. **Rare causes include:** viral infection (mumps) , hyperparathyroidism, corticosteroids & hypertriglyceridemia.

Pathology

-Pathogenesis

- There is premature activation of the pancreatic digestive enzymes while still in the pancreas → autodigestion of the gland.
- The exact mechanism of enzyme activation is not understood
- Inflammatory cytokines are produced and trigger an inflammatory cascade → SIRS
- The condition is like a major burn of the peritoneum, which produces abundant protein-rich exudate.
- The enzyme lipase which is released from the pancreas splits fat in the omentum and peritoneum producing glycerol and fatty acids → acids bind with calcium → insoluble calcium soap which manifests as white spots of fat necrosis
-
- The proteolytic activity of the liberated pancreatic enzymes produces vasoactive kinins and other chemical mediator → lead to haemodynamic and respiratory consequences

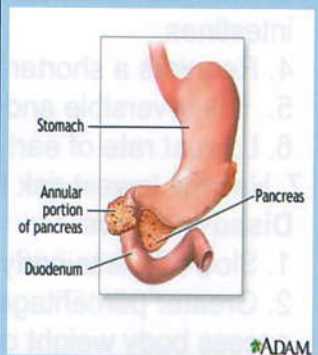
Gross picture

- The severity of pancreatic inflammation ranges from mild edema to hemorrhage → hemorrhagic ascites (hemorrhagic pancreatitis) and severe necrosis (necrotizing pancreatitis).
- White spots of fat in the peritoneum

Congenital anomalies

"Annular pancreas"

- Definition: a ring of pancreatic tissue that surrounds the second part of the duodenum → duodenal obstruction
- Type of patient: during the 1st year of life (½ of cases)
- Symptoms: Vomiting (main symptom)
- Signs: jaundice may be present.
- Investigations: contrast radiological studies.
- Treatment: duodeno-jejunostomy



Complications

Systemic complications (in form of multiple system failure)

1. Shock from:
 - a) Loss of plasma into the peritoneum and retroperitoneal
 - b) Loss of blood in hemorrhagic case
2. Adult respiratory distress syndrome → respiratory failure
3. Renal failure follows prolonged hypovolaemia.
4. Consumption coagulopathy.
5. Paralytic ileus and acute gastroduodenal stress ulcer & Hge
6. Tetany may occur because of hypocalcaemia.

Local complications

1. Pancreatic pseudocyst
2. Pancreatic abscess (4.5%).

Clinical picture

Symptoms

1. Upper abdominal pain (the main symptom in all cases)
Pain characterized by: a) Acute onset
b) Gradually increase in severity & becomes agonizing.
c) Commonly radiates to the back.
d) May follow a heavy meal or ingestion of alcohol.
2. Vomiting and retching are often present.

Signs

General signs

1. Fever and tachycardia are common.
2. Hypovolaemic shock in severe cases.
3. The patient commonly sits leaning forward as this positions partially relieves the pain.
4. A tinge of jaundice is sometimes observed

Local signs (less than expected from the severity of pain)

1. only mild tenderness and rigidity

This is explained by the position of the pancreas being a retroperitoneal organ → far from the sensitive parietal peritoneum.

2. blood-tinged exudate leak from the retroperitoneal space → Bruising around the umbilicus (**Cullen 's sign**), and in the loin (**Grey Turner's sign**)
(Rare late manifestations)
3. 2-3 weeks after the acute episode → upper abdominal swelling is sometimes observed (**pancreatic pseudocyst**).

- Some patients suffer from relapsing acute pancreatitis, enjoying relatively normal health between attacks
- Binding of acids with calcium is likely to produce hypocalcaemia in severe cases
- The occurrence of any of the system failures worsens the prognosis and if improperly treated, one system failure leads to another, and the chain ends in death.

-Pancreatitis begins when digestive enzymes become active inside the pancreas and start digesting it

-symptoms of acute pancreatitis include severe acute pain in upper abdomen, nausea & vomiting

-Shock commonly present with moderate tenderness & rigidity



Investigations

I. Laboratory

1. **Serum amylase** is usually elevated within a few hours to levels ≥ 1000 IU/dL (normal value is 100-300 IU/dL).

-It remains elevated for 2-3 days.

-The problem with serum amylase is that it is not specific for acute pancreatitis, it is elevated in other conditions as: perforated peptic ulcer, acute cholecystitis, intestinal obstruction and acute mesenteric ischemia. → enzyme level in these cases not exceed 500 IU/dL.

2. **Urinary amylase**

-used for diagnosis of patients who present after 2 days of the start of pain as the serum amylase is cleared out by the kidney

3. **Serum lipase.**

-Lipase has a slightly longer half-life.

- It is particularly useful in cases of delayed presentation.

-elevated lipase levels are more specific than amylase levels.

4. **Arterial blood gases**

- To detect cases who will need mechanical ventilation.

5. **Biochemical changes** → mild bilirubin elevation, hypocalcaemia, hypoproteinaemia, elevated blood urea & hyperglycemia.

6. **CBC** shows: leucocytosis and haematocrit is usually elevated because of fluid loss but lowered in hemorrhagic pancreatitis.

II. Imaging

1. **Plain X-ray of the abdomen**

- Shows a dilated short segment of the small intestine that is commonly referred to as a "**sentinel loop**".

-There may also be distension of the transverse colon and collapse of the descending colon (**colon cutoff sign**).

2. **Abdominal ultrasound**

-May show gall bladder stones.

-Stones impacted in the lower end of the CBD are usually not seen, yet their presence is indicated by dilatation of the biliary tree.

3. **CT scan with IV contrast** (very helpful in diagnosis)

- It reveals: enlargement of the pancreas, peri pancreatic edema and intraperitoneal fluid.

-Most important is its ability to detect pancreatic necrosis, which is diagnosed when a big part of the parenchyma is not enhanced after contrast injection

-The presence of necrosis = severe attack

- A CT scan should be performed at least 48 hours after the onset of symptoms to detect the extent of necrosis

4. **Magnetic resonance cholangiopancreatography (MRCP)**

-may be done if choledocholithiasis is suspected.

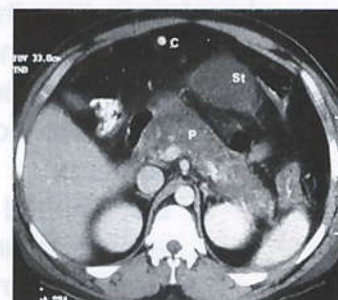
5. **ECG and the cardiac enzyme creatine phosphokinase (CPK)** are done to exclude myocardial infarction.

- Differential diagnosis from other causes of acute upper abdominal pain as:

- a) Perforated peptic ulcer.
- b) Acute cholecystitis and biliary colic.
- c) Acute mesenteric vascular occlusion.
- d) Leaking aortic aneurysm.
- e) Acute M.I.

All these causes of acute upper abdominal pain are serious and, if neglected, may be fatal

- The diagnosis of acute pancreatitis is mainly done by exclusion of other causes of acute upper abdominal pain.



Assessment of severity

Ranson's scoring system (asses' severity & prognosis)

On admission		Initial 48 hours	
Age	> 55 years	Haematocrit decrease	> 10%
White cell count	> 16000/uL	BUN elevation	>5mg/dl
Blood glucose	>200 mg/dl	Serum calcium	<8 mg/dl
Serum LDH	>350 IU/L	Arterial PO ₂	<60mmHg
AST	>250 IU/L	Base deficit	>4 mEq/L

Treatment

I- Conservative treatment (best)

- Aim: support of the different body systems
- it is easily remembered as the "R" regimen.

1. Relief of pain

- By pethidine (meperidine).
- This is combined with an atropine derivative to counteract the spasm of the sphincter of Oddi induced by opiates.

2. Replacement of the lost fluids

- By crystalloids and plasma.
- Blood is used in hemorrhagic cases.
- Is monitored by vital signs, urine output, CVP & haematocrit.
- Calcium is added to the infusions as required.

3. Respiratory support

- By oxygen mask or by mechanical ventilation in respiratory failure.

4. Rest of the pancreas and bowel

- By keeping the patient NPO (no oral intake).
- IV fluids are given for a few days until vomiting and nausea stop
- Nasogastric tube suction is sometimes needed.
- Severe prolonged cases require total parenteral nutrition

5. Resistance of infection

- By prophylactic antibiotics.
- The value of antibiotics is controversial, yet it may reduce the incidence of infection in cases of necrotizing pancreatitis.
- The preferred antibiotics are those of the carbapenem class, e.g. Imipenem/cilastatin or meropenem

6. Removal of an obstructing bile duct stone

- By ERCP and sphincterotomy in the acute attack is recommended if there is obstructive jaundice or cholangitis.
- Otherwise this intervention is not recommended during the attack

7. Reassessment when the symptoms completely resolve by ERCP.

- In biliary pancreatitis → cholecystectomy and removal of common duct stones, if not endoscopically removed, is performed within the same hospital admission.
- Alcoholic patients are advised to give up alcohol intake.

Patients with severe pancreatitis who should be treated in the ICU unit

II- Surgical Treatment

- indicated for:

- In severe necrotizing pancreatitis as detected by a CT scan. Necrotic tissue is excised and tube drains are left in the peritoneal cavity to allow postoperative lavage.
- Drainage of a pancreatic abscess → drained externally by a tube
- Persistent pseudocyst that does not resolve in 6 weeks → internally drained to the stomach or to a jejunal loop

Chronic pancreatitis

Clinical picture

- Epigastric pain that radiates to the back.
 - It may be mild or severe, and continuous or interrupted.
 - Commonly causes drug addiction for analgesics.
- Malabsorption (from loss of enzymes)
 - Produces steatorrhea in 1/3 of cases with loss of weight & weakness.
 - Stools typically are soft, greasy & foul smelling.
- Diabetes mellitus (develops in extensive pancreatic damage).
 - It occurs in 1/3 of patients.
- few physical signs:
 - Malnutrition and a tinge of jaundice (in 3% of cases)
 - There is epigastric tenderness

Investigations

- Laboratory** (no laboratory test specific for chronic pancreatitis)
 - Steatorrhea is assessed by measuring faecal fat excretion
 - Lund test.
 - Secretin & pancreozymin stimulation → show reduced pancreatic secretion.
 - Glucose tolerance test.
 - Serum bilirubin and other liver function tests

II-Imaging

- Plain x-ray : may show calcification or stones.
- Ultrasound and CT scan detect pancreatic enlargement and show associated biliary pathology.
- Endoscopic retrograde cholangiopancreatography (ERCP)
 - is of great value and performed if operation is considered.
 - The architecture of the pancreatic duct is revealed.
 - Common features include strictures, stones, cysts, or alternating strictures and dilatations of the pancreatic duct, which give an appearance known as the "**chain of lakes**".
 - Pure pancreatic juice can be obtained for cytology to exclude malignancy
- Magnetic resonance cholangiopancreatography (MRCP)
 - provides information on the pancreatic ductal system.
 - is relatively safe, reasonably accurate, noninvasive, fast and very useful in planning surgical or endoscopic intervention.

-Incidence:

Rare in Egypt

-Etiology:

- Over consumption of alcohol (**the main cause**)
- Stone in the lower end of CBD (**2nd common**)

-Pathology:

- There is one or multiple strictures of the pancreatic duct.
- Irreversible replacement of the acinar cells with fibrous tissue
- Gradual loss of both exocrine and endocrine functions.
- In a few cases inflammation and fibrosis of the head of the pancreas → obstruct the lower part of CBD → obstructive jaundice



5) Endoscopic ultrasonography

- The **best test** for imaging the pancreas
- Requires a highly skilled gastroenterologist.
- The most predictive feature is the presence of stones



Treatment

Abstinence from alcohol intake is essential.

I-Conservative treatment (for the majority of cases)

- 1) Control of pain by non-opiate analgesics(to avoid addiction)
- 2) Low fat diet with oral supplementation of fat soluble vitamins
- 3) Correction of malabsorption by intake of pancreatic enzyme tablets with meals.
- 4) Control of diabetes by insulin therapy.

II- Surgery

- Indicated for: persistent uncontrolled pain.
- Options are :

- a) Resection of a part of the pancreas is done when it is severely damaged and the rest of the organ is relatively healthy.
- b) Drainage of a dilated pancreatic duct by pancreaticojejunostomy.
- c) Coeliac block interrupts the pain pathways from the pancreas and relieves pain of chronic pancreatitis.

Therapeutic value of ERCP:

-Endoscopic division of the sphincter of Oddi (sphincterotomy) is done in cases of obstruction of the duct opening with stone or with stenosis of the duodenal papilla.

Pancreatic pseudocyst

Pathology

1) Nature.

-This is a collection of pancreatic secretion & inflammatory exudate within a lining of inflammatory tissue rather than epithelium so it is called a pseudocyst (false cyst).

2) Etiology

a) develops in 10% of acute pancreatitis usually after 2-3 weeks

b) Pancreatic trauma.

c) Chronic pancreatitis.

3) Site

- In the lesser sac between the pancreas and the stomach.

4) Complications: infection, hemorrhage & rupture.

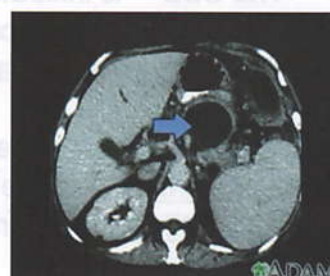


Clinical picture

1. A small pseudocyst is painless and may be discovered by follow up ultrasonography.
2. A large cyst causes discomfort and manifests as an upper abdominal swelling that may reach the size of a melon

Investigations

- 1) Endoscopic ultrasound and aspiration of fluid → will reveal high level of amylase.
- 2) CT scan (most accurate)

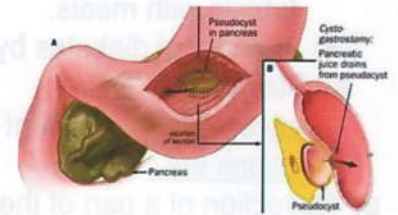


Treatment

- Most of these cysts resolve spontaneously
- clinical and ultrasound follow-up is needed
- A persistent pseudocyst for 6 weeks → is surgically drained.
- Waiting for 6 weeks to allow the development of a strong cyst wall that can hold sutures.
- The cyst is internally drained to the stomach (Cystogastrostomy) or to a jejunal loop (cystojejunostomy)
- Is done by making an incision in the anterior wall of the stomach, then incising the posterior gastric wall and the cyst wall which are adherent to each other.
- A continuous interlocking suture binds the posterior gastric wall to the cyst wall securing homeostasis and making an opening between the cyst and the stomach → the cyst gradually disappears

Endoscopic drainage of the cyst to the stomach is feasible in the presence of resources and experience

Cystogastrostomy



Pancreatic neoplasms

Exocrine (common)		Endocrine (rare)	
Benign	Adenoma	Alpha cells	Glucagonoma
		Beta cells	Insulinoma
Malignant	adenocarcinoma	Delta cells	Somatostatinoma
		Non beta cells	gastrinoma

Pancreatic adenocarcinoma

Pathology

1) Gross picture

- is infiltrating, hard and irregular.

2) Site

-In the head : (60 %) → 2/3 in the head proper & 1/3 in periampullary (ampulla of Vater , duodenal papilla or lower end of CBD)

-In body and tail : (40 %)

3) Microscopic picture.

- is an adenocarcinoma arising from the ductal system
- Cystadenocarcinoma is rare.

4) Spread

a) Direct to :

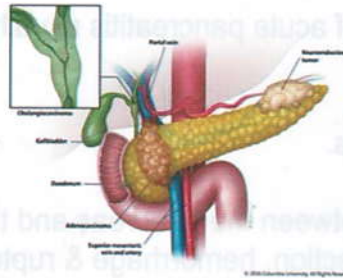
- The CBD → is infiltrated early in pancreatic head cancer → obstructive jaundice.
- The duodenum, portal vein, and inferior vena cava

b) Lymphatic

- To adjacent lymph nodes & to nodes in the porta hepatis.

c) Blood spread → to the liver (mainly)

d) Transperitoneal → peritoneal seeding and ascities



-Incidence:

1. lies between 55 & 70 years of age
2. Affects males more than females
3. The prognosis is poor → The 5-year survival rate is about 5%

-Etiology:

1. The exact etiology is unknown.
2. Factors that increase the risk: smoking, obesity, chronic pancreatitis & diabetes

Clinical picture

I- Carcinoma of the head.

- 1) obstructive jaundice and pruritus.
- Is painless jaundice (sometimes painful)
- Progressive (may be temporarily if an obstructing periampullary carcinoma sloughs)
- Deep olive green -pruritis
- 2) The liver enlarged (because it is engorged with bile)
- 3) The gall bladder is distended in 1/2 the cases.
- 4) Anorexia and loss of weight are marked.

II- Carcinoma of the body and tail (manifest late)

-The manifestations are nonspecific

-Include:

- 1) Anorexia, loss of weight, and weakness.
- 2) Epigastric pain radiating to the back (a late symptom).
- 3) Migrating inflammation of the superficial veins
(Thrombophlebitis migrans→ accompany visceral malignancies)
- 4) The liver may be enlarged (from metastases) and ascities

-Endocrine neoplasms that arise from APUD cells, are termed apudomas

-They are interesting in regard to their metabolic consequences and the Possibility of associated APUD neoplasms in other organs , that is known as multiple endocrine neoplasia (MEN) syndrome

- **Differential diagnosis:**

- 1) Calcular obstructive jaundice
- 2) Chronic pancreatitis.
- 3) Other causes of jaundice, e.g., viral hepatitis

	Calcular obstructive jaundice	Malignant obstructive jaundice
Peak age incidence	Middle age	Elderly
Gender	More in females	More in males
Duration of jaundice	May be long	Short
Course of jaundice	Intermittent	Progressive
Depth of jaundice	Moderate	May be very deep
Abdominal pain	Colicky, more in epigastrium and right hypochondrium	May be absent→ is dull aching and referred to back
Anorexia & weight loss	Absent	Marked
Fever	May be present	Usually absent
Gall bladder	Usually impalpable (Courvoisier's law)	Commonly palpable
Ultrasound	Usually fibrosed gall bladder with stones	Marked distended thin-walled gall bladder
CT scan	Gallstones	Head neoplasm is usually seen
ERCP	Stone in CBD	Abrupt termination or failure of cannulation of CBD

I-laboratory

1) Liver function tests

- A) Bilirubin : elevated (mainly direct bilirubin),
- B) alkaline phosphates → high
- C) prothrombin concentration → low (from defective absorption of vitamin K due to the lack of bile salts in the intestine)

2) Tumour markers

- a) CA 19-9 (useful method of confirmation and in follow-up tt)
- b) Carcinoembryonic antigen (CEA)

- (CEA) is a tumour marker for GIT carcinomas in general, is elevated in only 40% - 50% of patients with pancreatic cancer.

-Biopsy is not needed if clinical evidence, elevated CA 19-9 and a hypodense lesion on triphasic CT are all present.
-When CT appearance is atypical, needle biopsy is considered.
-A positive cytology justifies resection of operable cases. On the other hand a negative result cannot rule out malignancy

II-imaging

1) Abdominal ultrasound

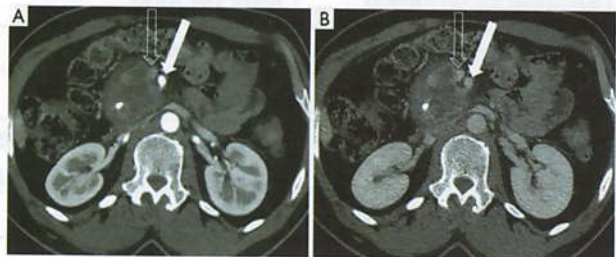
- **The 1st imaging** done when obstructive jaundice suspected.
- May fail to show the pancreatic carcinoma because of the overlying colonic gases
- The examination is useful to:
 - a) Document the case as extra hepatic obstructive jaundice by showing dilated extra and intrahepatic bile ducts.
 - b) Sometimes finds gallstones(the cause of obstruction)
 - c) Liver metastases are readily diagnosed by ultrasound.

2) Endoscopic ultrasound

- shows the relation of the tumour to the blood vessels and allows taking a biopsy

3) CT scan

- ideally should be triphasic, spiral CT with thin cuts (3 mm)
- It shows the tumour, its local extent, and liver metastases
- The tumour classically appears as a hypodense lesion
- Important to look for invasion of the superior mesenteric vessels and the portal vein.
- It allows targeting percutaneous FNAC of the lesion.



4) Endoscopic retrograde

cholangiopancreatography (ERCP) or percutaneous transhepatic cholangiography (PTC)

- Exact delineation of the site of obstruction.
- Differentiate malignant from stone obstruction of the bile duct
- Placing a stent in the bile duct allows drainage of bile to relieve jaundice either as a preparation for surgery, or permanently in very ill inoperable cases.



Treatment

I- Patients who are unfit for surgery

- jaundice can be treated by an endoscopic stent passed from the duodenum into the common bile duct and then past the block to the dilated area above → drain bile into the duodenum
- Self-expanding metal stents are better than plastic stents.

II- Patients who are fit for surgery

- Surgery is the best mode of treatment.

A- Possible risks of surgery on obstructive jaundice patients:

- 1- Excessive hemorrhage resulting from hypoprothrombinaemia due to lack of absorption of vitamin K.
- 2- Septicaemia caused by bacteria migrating from the bowel to the blood stream.
- 3- Renal and hepatic failure consequent upon the septicaemia.

B- Preoperative preparation (to avoid the mentioned risks)

- 1- Parenteral vitamin K administration at a dose of 10 mg IV bid (oral vitamin K is not absorbed)
- 2- Administration of oral bile salts for 2 days before surgery → prevents migration of bacteria to the blood stream.
- 3- Prophylactic parenteral antibiotics against Gram -ve bacilli are given before & continued during and after the operation.
- 4- The patient is kept adequately hydrated.

- If the bilirubin is above **15 mg/dL** → give mannitol before, during & after surgery (This is an osmotic diuretic that is administered intravenously and helps reduce the incidence of renal failure)
- 5- Sugar-rich food helps the liver build up glycogen to stand the stress

C- Operation

- I- **-for operable cases (10 -15% of cases) we do pancreaticoduodenectomy** → known as Whipple operation

- The following structures are removed:
 - 1) The head and neck of the pancreas.
 - 2) The whole duodenum(as it shares the same blood supply as the head of pancreas)
 - 3) The antrum of the stomach.
 - 4) The gall bladder and the common bile duct.
- The operation ends by performing the following anastomosis:
 - 1) The hepatic duct to the jejunum.
 - 2) The pancreatic duct to the jejunum or stomach.
 - 3) The stomach to the jejunum.
- The mortality rate of operation approach 10%.
- The 5-year survival after resection is poor but is better with periampullary carcinoma (30%) than carcinoma of the head proper (0-5%)

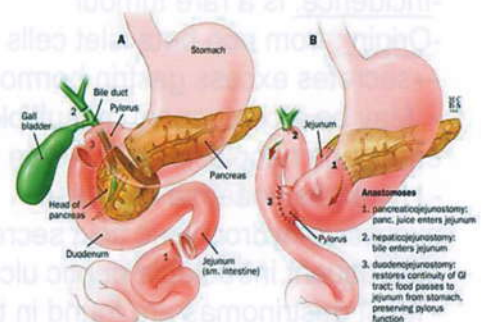
Operable
(Potentially curable)
(Represent **10-15%**) are those who have neither liver nor peritoneal Deposits & are not fixed to the hepatic or celiac artery.

- These cases deserve a radical operation, which is **pancreaticoduodenectomy**, known as **Whipple operation**

- Adjuvant chemotherapy and/or radiotherapy may be given after resectional surgery or before operation (neoadjuvant therapy).

- Inoperable cases are that either have:

- 1) liver or peritoneal metastases
- 2) involvement of coeliac lymph nodes
- 3) invasion of the SMA



For inoperable (incurable) = majority of cases

- Drainage of bile to the intestine either by hepaticojejunostomy or cholecystojejunostomy to avoid the risk of future duodenal obstruction by the growing tumour (some surgeons routinely add a gastrojejunostomy)



Insulinoma

Clinical picture

- **Type of patient** : adults under 40 years who are usually overweight due to over eating.

- Bizarre behavior and unconscious episodes.

- Some patients are misdiagnosed as having a psychiatric illness.

- Palpitation, nervousness and other symptoms of sympathetic hyperactivity.

- **Whipple's triad**:

1) Symptoms of hypoglycemia (sweating , anxiety ,tremor ,confusion and coma) that are precipitated by fasting or exercise.

2) During the attack → blood glucose level <45 mg/dL.

3) The symptoms are relieved by glucose.

Investigations

I- Laboratory

1- Low blood glucose level

2- High insulin level in the blood.

II- Imaging

- Localizing the tumor by endoscopic ultrasound and CTscan.

Treatment

The part of the pancreas that contains the tumour is resected

Gastrinoma (Zollinger-Ellison syndrome)

Pathology

- **Incidence**: is a rare tumour

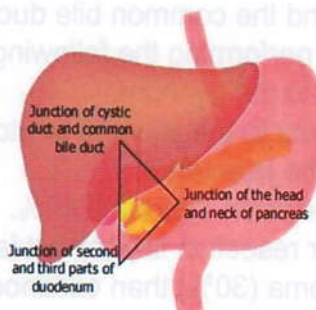
- **Origin** : from non beta islet cells

→ secretes excess gastrin hormone.

- May be a component of multiple endocrine neoplasia (MEN) type I

- Hypergastrinaemia results in excessive hydrochloric acid secretion → consequent intractable peptic ulceration.

- Most gastrinomas are found in the gastrinoma triangle



-Pathology:

1. **Origin**: a tumour of the beta cells of the islets of Langerhans.

2. Is usually benign, few cases are malignant.

3. **Site** : commonly found in the body and tail(rarely multicentric)

4. Some insulinomas are part of multiple endocrine neoplasia (MEN) type I with associated anterior pituitary adenomas and parathyroid hyperplasia.

5. The specific feature is the secretion of excess insulin irrespective of the blood glucose level→ hypoglycaemia

- Intraoperative ultrasound is a very useful localization method

Clinical picture

- Abdominal pain (caused by peptic ulceration→ may bleed or perforate)
- The diagnosis of Zollinger-Ellison syndrome is suspected in the following situations:
 - 1) Ulcers resistant to treatment with H₂receptor antagonists.
 - 2) Recurrent ulcers.
 - 3) Multiple ulcers and ulcers in unusual sites as the distal duodenum and proximal jejunum.
 - 4) Peptic ulcers at the extremes of age.
 - 5) Diarrhea (present in 1/3 of cases)

• In contrast to Insulinoma, 60% of gastrinomas are malignant

Investigations

- 1- Upper GI endoscopy & barium meal examination show the ulcers.
- 2- Radioimmunoassay estimation of serum gastrin.
 - The normal level is usually < 150 ng/L.
 - Most cases show levels > 500 ng/L.
 - Some patients have intermediate levels between 200-500 ng/L, such cases show doubling of the serum levels when stimulated with intravenous calcium or secretin.
- 3- Localization of the tumour by CT scan.
- 4- Endoscopic ultrasound.
- 5- ¹¹¹In octreotide scintigraphy

Treatment

- The ideal treatment is to excise the part of the pancreas containing the tumour.
- Only 20% of tumours are suitable for resection(because of multiplicity and poor localization)
- Commonly tumour resection is not possible and total gastrectomy is done to completely abolish acid secretion.
- A less effective alternative is prolonged intensive therapy with proton pump inhibitors

Intraoperative ultrasound is very useful in localization

Peritoneum, Omentum and Mesentery

Generalized septic peritonitis

Etiology

Causative organisms include:

- 1- E. coli
- 2- Aerobic and anaerobic streptococci
- 3- Bacteroides
- 4- pneumococci.

Sources of infection

A- Local spread (the commonest route)

1- **Infected organs** as: appendicitis, cholecystitis or diverticulitis.

2- **Leaking organs** as: perforated peptic ulcer, perforated typhoid ulcer, leaking anastomosis, ruptured gut or extravasated urine.

B- Direct entry through an operative or traumatic wound.

C- Blood spread may occur in septicaemia and pyaemia (rare)

Pathology

Fate:

1- Resolution

-The peritoneum has great resistance to infection.

-If source of infection is controlled or removed → peritonitis resolves

2- Localization

 (abscess formation).

- If complete resolution fails, pus localizes → an abscess.

- Localization may occur either:

a) Around the 1st focus (as in acute appendicitis or cholecystitis) by adhesions of intestine and omentum around the inflamed organ

b) In one of the anatomical compartments of the peritoneal cavity to form a remote abscess as: a subphrenic, iliac or pelvic abscess

3- Flaring up

 → diffuse (generalized) peritonitis

Predisposing factors include:

a) High virulence of the organisms.

b) Sudden perforation of a hollow viscus → not allows the defensive mechanisms to localize the source of infection.

c) Persistent source of infection.

d) Stimulation of peristalsis by eating, enemas or purgatives.

e) Immunosuppression as in diabetes mellitus, AIDS & patients receiving corticosteroids or chemotherapy.

f) In children because the greater omentum is small in size & not well developed (has an important role in localization)

4- Septic shock

 → multiple organ failure (MOF) & death.

Clinical picture

Symptoms

1- Pain: -Character: dull aching & persistent

- Increases by: movements or coughing.

-The site of maximum pain: at the site of the original lesion.

2- Symptoms of paralytic ileus

a) Vomiting → at first gastric contents then becomes bilious.

-In long-standing cases, the vomitus becomes feculent.

b) Abdominal distension & absolute constipation.

-This is a life-threatening condition.

-Gross changes

1- Fibrin deposition → inflamed areas become opaque & adhere → a pyogenic membrane

2- Excess purulent exudate accumulates.

3- At first: Paralytic ileus

-Later: fibrinous adhesions → mechanical I.O.

- **Fate** depends on: i) the virulence of the organisms
ii) the efficiency of treatment
iii) the body resistance.



Primary peritonitis

Incidence:

-uncommon form of septic peritonitis

- Usually affects female children

Etiology:

- No apparent intraperitoneal pathology.

-main organisms → Pneumococci & Streptococci

-route → enter the peritoneum through the fallopian tubes.

Signs

A-General:

- The patient looks distressed, toxic, lies still in bed avoiding any movements
- There is pyrexia & tachycardia.
- In neglected cases → presents by sunken eyes, anxious look (**facies Hippocratica**) & clinical picture of septic shock

B-Local abdominal examination:

- **Palpation:** generalized tenderness, rebound tenderness & rigidity
- **Auscultation** → absent bowel sounds (due to paralytic ileus)

Investigations

A-Laboratory

- CBC → Polymorphonuclear leucocytosis with shift to left

B-Imaging

1-Plain abdominal x-ray

- Shows paralytic ileus with distension of the small & large bowel.
- It may determine 1^{ry} lesion as:
with perforated peptic ulcer → air under the diaphragm.

2-Abdominal ultrasound or CT

- Reveals free fluid in the peritoneal cavity.
- Guided peritoneal aspiration → determines the nature of fluid and allows bacterial culture & sensitivity



Treatment

Preoperative preparation

1-NG tube → to deflate the stomach & bowels → prevent vomiting during induction of anaesthesia.

2- IV fluids as saline or Ringer's solution to correct hypovolaemia.

3- **Antibiotics:** a combination of ampicillin, an aminoglycoside & metronidazole → cover all aerobic and anaerobic organisms.

- May be changed after results of pus culture & sensitivity.

4-**Analgesics**

5- A Foley catheter: to check urine output & adequacy of fluid replacement.

Operation

Exploratory laparotomy done when general condition is satisfactory

1- General anaesthesia.

2- Long midline incision

- Pus is sampled for culture and sensitivity and is suctioned out.

3-Deal with the 1^{ry} lesion to stop further contamination

- Ex: a perforated appendix → removed

A perforated peptic ulcer → closed by an omental patch.

4-**Peritoneal toilet** by copious irrigation with sterile saline

5-**Peritoneal drainage** may be done.

- The drains come out through separate stabs.

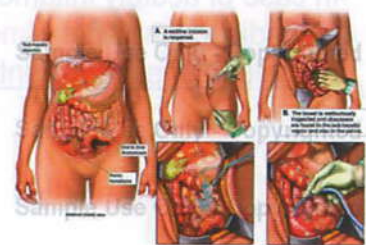
-At first→ patient presents by the clinical picture of the original cause as acute appendicitis or perforated duodenal ulcer, later when the condition is fully established → difficult to tell original cause

- The site of maximum tenderness & rigidity usually indicates the source of infection

- The treatment of pus anywhere in the body is **Drainage.**
- Acute generalized peritonitis is a surgical emergency
- Drainage of the subcutaneous space is also useful as the wound is very likely to get infected



Exploratory Laparotomy and Small Bowel Resection



Localized septic peritonitis (intraperitoneal abscess)

- An intraperitoneal abscess = the defensive mechanisms successfully localized the source of infection.
- These protective mechanisms include:
 - 1-the omentum (**abdominal policeman**)
 - 2- Matting of loops of bowel around the source of infection.
- The common sites of intraperitoneal abscesses are:
 1. The iliac fossa
 2. pelvis
 3. subdiaphragmatic space

Iliac abscess

Etiology

A-On the right side

- 1- Acute appendicitis.
- 2- Perforated duodenal ulcer

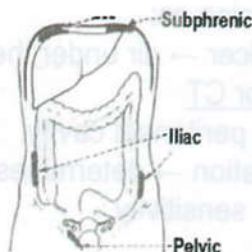
The exudate trickles through the right paracolic gutter

B- On the left side

- 1- Perforated diverticulitis.
- 2- Perforation of carcinoma of the sigmoid colon.

C-On either side

- 1- Spread from the female genital organs.
- 2- 2nd to generalized peritonitis (localization)



Clinical picture

Symptoms:

- General: hectic fever
- Local: 1-Iliac pain 2-vomiting.

Signs: 1-Iliac tenderness & rigidity. 2- Tender mass may be felt.

Investigations

A-Laboratory

CBC → polymorphonuclear leucocytosis

B-Imaging

Ultrasound examination:

- determines the site & size of the abscess
- permits guided: a) Aspiration for bacterial culture
- b) Drainage as a form of treatment

Treatment

1-Drainage of pus (the cornerstone) either by:

Surgery or guided percutaneous placement of a catheter

2- Controlling the cause.

-In case of acutely inflamed appendix → not removed → an interval appendectomy after **6 months** (much easier & safer)

3- The use of effective antibiotics.

Pelvic abscess

-Definition:

Is a collection of pus in the recto-vesical pouch or Douglas pouch

-Causes:

1- Acute appendicitis.

2- Localization of resolving diffuse peritonitis.

3- Pelvic inflammatory disease in females

- C/P:

A-Symptoms

General: Hectic fever & toxemia.

Local: 1-Deep pelvic pain.

2. Diarrhoea and tenesmus (due to rectal irritation)
3. Burning micturition & frequency (due to bladder irritation)
4. If neglected → bursts through the rectum or the vagina

B-Signs

DRE → fullness & tenderness in front of rectum

-Treatment:

Drainage according to the site of pointing:

- a) If in rectum → transrectal drainage.
- b) If in vagina → drained through the posterior fornix
- c) If in the suprapubic region → percutaneous ultrasound guided

Subphrenic abscess

Etiology

- 1- Residual pus collection following generalized peritonitis.
- 2- Perforated viscera as appendix, gall bladder or peptic ulcer.

Clinical picture

A-Symptoms

General: 1- fever 2- persistent hiccough

Local:

- 1- May be totally absent
- 2- Slight pain
 - **Site:** in epigastrium
 - **referred to :** the shoulder (due to phrenic nerve irritation)

B-Signs

General: 1- Hectic fever. 2- Tachycardia.
3- Severe toxemia with anorexia, vomiting & sweating.

Local

- a) **Inspection:** Impaired chest movement on the affected side.
- b) **Palpation:**
 - i) Tenderness may be present over the lower ribs & intercostal spaces or below the costal margin.
 - ii) There may be swelling and rigidity of the upper abdomen.
- c) **Auscultation :** Impaired air entry & crepitations over the lung base of the affected side

Investigations

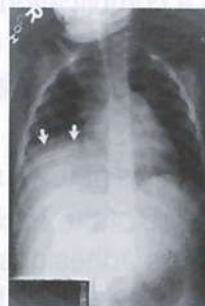
A-Laboratory

CBC → polymorphonuclear leucocytosis with shift to the left

B-Imaging

1- Plain chest X-ray:

- a) Elevated fixed diaphragm.
- b) Obliteration of costophrenic space by a minimal pleural effusion
- c) Gas under the diaphragm or air fluid level (when the cause is a perforated viscus or there is infection with gas forming organisms)



2- Abdominal ultrasound and CT

- Localize the abscess.
- Allow guided aspiration for bacterial culture or guided drainage



Anatomy of subphrenic space:

The subphrenic region is portion of the abdominal cavity which extends from:
Above → the diaphragm
Below → to the transverse colon & mesocolon.

-The region is divided by the liver into:

1-suprahepatic compartment → divided by falciform Ligament into: right and left parts.

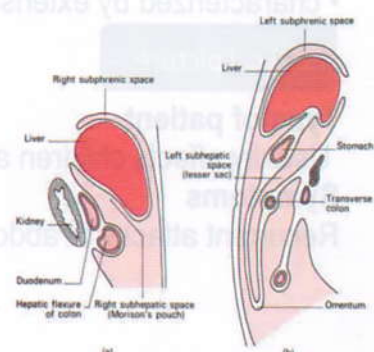
2- Infrahepatic compartment.

-The right infra hepatic space (**hepatorenal pouch of Morrison**):

-Is the most dependent part of the abdomen when a person is lying down → the preferred site for pus to collect.

- Above and in front are: the liver and gallbladder

- Below and behind are: the upper pole of the RT kidney, RT adrenal and 2nd part of the duodenum



Treatment

1- Conservative treatment

Indication: early cases

Method: by antibiotics

2- Percutaneous aspiration

Indication: failure of conservative treatment

Method: placement of a percutaneous catheter under ultrasound or CT guidance.

3- Open drainage by surgery

Thick pus and a multilocular abscess

-subphrenic abscess is suspected if there are general manifestations of pus but cannot find the source particularly after abdominal surgery or with inflammation of an abdominal organ
-Pus somewhere, Pus nowhere else, Pus under the diaphragm

Tuberculous peritonitis

Etiology

Is always 2^{ry} to a tuberculous focus elsewhere that reaches the Peritoneum through:

1- Direct spread as

- a) tuberculous salpingitis (**the commonest cause**)
- b) TB lymphadenitis
- c) TB enteritis.

2- Blood spread e.g. pulmonary tuberculosis.

3- Lymphatic spread e.g. from pleura or bowel.

Pathology

1- Ascitic type (**the commonest type**)

- The peritoneum is studded with tubercles.
- There is copious amount of straw colored ascites.
- The greater omentum is thickened, fibrosed & frequently rolled up forming a sausage shaped mass above the umbilicus.

2- Caseous type (purulent form)

- It affects young females as a complication of TB salpingitis.
- The peritoneum is studded with tubercles and is thickened
- Multiple collections of caseous material are present among the adherent bowel and omentum.
- Cold abscess may form & may point to → sinuses or fistulae.

3- Encysted type (localized form of the ascitic type)

- The fluid is encysted by fibrous adhesions and coils of bowel.
- It produces an intra-abdominal cyst (may be mistaken for ovarian and mesenteric cysts)

4- Adhesive type (fibrous or plastic form)

- characterized by extensive peritoneal adhesions → I.O.

Clinical picture

Type of patient

Usually affects children and young adults of both sexes.

Symptoms

Recurrent attacks of abdominal pain, vomiting & distension.

Signs

General:

TB toxæmia → night fever, night sweats, anorexia & wasting.

Local:

i) Palpation:

1- The abdomen is felt doughy with multiple palpable swellings (may be LNs or omentum)

2- Sausage shaped mass felt above the umbilicus (Rolled omentum)

3- Tenderness may be present.

ii) **Percussion:** Ascites is a common finding.

iii) **PV:** may reveal a tubo-ovarian mass.

Investigations

A-Laboratory

1- **CBC:** may reveal anaemia, lymphocytosis & raised ESR.

2- **Tuberculin test:** highly positive.

B-Imaging

1- Plain chest X-ray.

2- Abdominal ultrasound

May reveal loculated or free ascites or multiple swellings due to caseous collections of pus among adherent loops of intestine

C-Instrumental

1- Abdominal tapping

-indication: performed in the ascitic type.

-The aspirated fluid is:

a) Clear & straw colored

b) Specific gravity >1020

c) Very rich in lymphocytes.

- To demonstrate TB bacilli → culture or PCR (better & necessary).

2- Laparoscopy with biopsy (the gold standard for diagnosis)

- Direct visualization of the characteristic tubercles & permits biopsy.

- A solid proof of the diagnosis → antituberculous drugs can be taken

3- Exploration (seldom needed)

It is difficult to detect TB bacilli by direct films

Treatment

A-Medical (main treatment)

- At least two antituberculous drugs (isonicotinic acid hydrazide & rimactane) are prescribed for at least one year.

B-Operation

-rarely indicated e.g. I.O.

Ascites

Definition

A pathological accumulation of fluid in the peritoneal cavity

- It can be recognized clinically when the amount of fluid > 1500 ml.

Etiology

A- General causes (generalized tendency for edema formation)

- 1- Liver diseases, e.g. liver cirrhosis.
- 2- Cardiac diseases, e.g. heart failure.
- 3- Renal diseases, e.g. nephrotic syndrome.
- 4- Nutritional defects, e.g. low serum albumin.

B- Local causes (exudate)

- 1- T.B peritonitis (acute miliary, ascitic type).
- 2- Malignant ascites (Usually 2^{ry} to carcinoma of abdominal organs)
- 3- Chylous ascites.
- 4- Pancreatic ascites.

C- Rare causes

- 1- Meigs' syndrome (due to ovarian fibroma).
- 2- Pseudomyxoma peritonei.

Differences between the main causes of ascites

	Cirrhotic ascites	Malignant ascites	Tuberculous ascites
Age	Any age	Usually elderly May be young with ovarian cancer	Usually young
History	jaundice or haematemesis	short history There may be symptoms of 1 ^{ry} tumour	Long history of low-grade fever, abdominal pain & weight loss
General examination	Features of liver insufficiency	Probably distant metastases	Low-grade fever & cachexia
Abdominal examination	Hepatomegaly and/or splenomegaly	Multiple hard abdominal masses may be palpable	Doughy mass may be palpable
Liver function tests	Poor	Normal, but may be impaired with liver metastases	Normal
Ultrasound	Cirrhotic liver & Splenomegaly	Abdominal masses may be detected	Abdominal masses may be detected
Tapping	-Transudate -Specific gravity <1020	-Exudate -Specific gravity > 1020 -Serosanguinous -Cytology may show malignant cells	-Straw colored exudates -Specific gravity >1020 -Culture or PCR diagnostic
Treatment	-medical treatment. -Peritoneo-venous shunt	-Tapping -Local hyperthermic chemotherapy	Antituberculous drugs
Prognosis	Bad	Very bad	Good

Peritoneal tumours

A- Peritoneal carcinomatosis

Pathology

Origin

- 1- Caused by implantation of secondaries on peritoneal surfaces.
- 2- Is a terminal event in many cases of carcinoma of the stomach, colon, breast, ovary & other abdominal organs.

Gross picture

- 1-The peritoneum (both visceral & parietal) is studded with malignant nodules.
- 2-The peritoneal cavity contains ascitic fluid which is usually blood stained.

Treatment

The condition used to be considered incurable and terminal

▪ Recent introduction of combined:

- a) Excision of the primary tumour
- b) Peritonectomy
- c) Hyperthermic intraperitoneal chemotherapy.

Has improved
5-year survival

- Peritonectomy entails excision of the visceral & parietal peritoneum + the omentum.

B-Pseudomyxoma peritonei

Etiology

- 1- Rupture of a pseudomucinous cyst of the ovary.
- 2- Rupture of a mucoid carcinoma of the appendix

Pathology

- 1-The abdomen is filled with yellow jelly-like material.
- 2-A low grade inflammation occurs → fibrinous or fibrous adhesions.

Clinical picture

Abdominal examination shows:

- 1- Distended abdomen without shifting dullness.
- 2- Multiple firm masses are palpable

Treatment

-The recent introduction of combined:

- a) excision of the primary tumour
- b) peritonectomy
- c) hyperthermic intraperitoneal chemotherapy

Has partially improved the 5-year survival of this aggressive malignancy

C-Mesothelioma

- Is a 1st neoplasm of the peritoneum (in contrast to the above mentioned 2 tumors)
- Is also known to affect the pleura.
- Incidence: rare
- Cause: suspected to be caused by exposure to asbestos.
- Types & presentation: either
 - a) Diffuse → ascites
 - b) Localized & bulky → an abdominal mass.

Torsion of the omentum

Clinical picture

- 1- Sudden abdominal pain.
- 2- The abdomen is rigid and tender.
- 3- The twisted omentum may be palpable

Treatment

Excision of the strangulated omentum

-Torsion of the omentum is predisposed to by adhesions of the omentum to a hernia or an old focus of infection
- Usually diagnosed only after exploration.

Mesenteric cysts

Types

1- Chylolymphatic cyst (The commonest)

-Etiology: retention cyst caused by obstruction of lymphatic drainage.

-Pathology: The cyst is thin walled, lined with endothelium & contains lymph (chyle).

-Its blood supply is separate from that of the related intestinal loop.

-Treatment: is enucleation (leaving the related intestine intact)

2- Enterogenous cyst

-Cause: represents incompletely duplicated intestine.

-Pathology:

Thick walled, lined with intestinal mucosa & contains mucous.

- Its blood supply is derived from the same vessels of the related intestinal loop

-Treatment: is by excision with resection anastomosis of the related loop of intestine.

3- Teratomatous dermoid cyst

4- Hydatid cyst

Treatment is by enucleation

Clinical picture

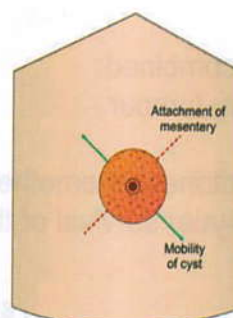
Type of patient: more in children and young adults

Symptoms

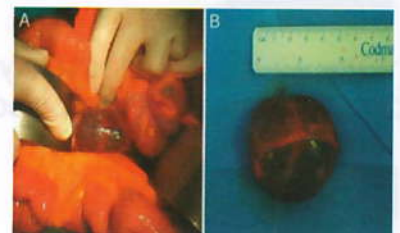
- 1- Abdominal swelling or dyspepsia (usual complaint)
- 2- Pain (usually absent)

Signs → Tillaux triad

- 1- The cyst is near the umbilicus
- 2- It moves across but not along the root of mesentery
- 3- The cyst is dull on percussion but there is a zone of resonance around & over it → due to the presence loops of bowel surrounding & in front of it.



Intraoperative pic of the Enterogenous cyst.



Large abdominal cysts

- 1- Pancreatic pseudocyst → in upper abdomen
- 2- Mesenteric cyst → mid abdomen, characteristic mobility
- 3- Ovarian cyst → pelvi-abdominal
- 4- Hydronephrosis → extends to renal angle

Mesenteric lymphadenitis

Acute non-specific mesenteric adenitis

Incidence

One of the commonest causes of acute abdominal pain in children

Etiology

Exact cause is unknown but may follow viral respiratory infection

Clinical picture

Type of patient

- It affects children and is unusual after puberty

Symptoms

1- Upper abdominal pain which localizes to the right side.

- Character: colicky and severe

- Duration: lasts for a short time.

2- There may be nausea, vomiting, malaise, anorexia & fever.

Signs

1- Voluntary abdominal guarding but no rigidity.

2- Abdominal tenderness (marked along the line of mesentery) and sometimes rebound tenderness.

3- DRE → tenderness may be elicited.

4- Shifting tenderness.

- This is a valuable sign for differentiation from appendicitis.

- After laying the patient on the left side for a few minutes → the point of maximum tenderness shifts towards the middle line.

-Pathology:

Macroscopically

1- The disease involves primarily the ileocaecal LNs → enlarged.

2- A small amount of clear serous fluid within the peritoneal cavity.

Microscopically

1- the nodes show a pattern of reactive hyperplasia.

2- Culture is usually negative

- The clinical features are close to those of acute appendicitis.
- It is not easy to differentiate the two conditions and whenever in doubt → it is much safer to operate on the patient than to miss an inflamed appendix in a child.

	Acute appendicitis	Non-specific mesenteric lymphadenitis
Course of pain	Continuous	Periods of complete freedom of pain
Point of maximum tenderness	McBurney's point	Above and medial to McBurney's point
Rovsing's sign	Positive	Negative
Shifting tenderness	Absent	Present

Treatment

1- Usually these patients are operated upon with a provisional diagnosis of acute appendicitis.

2- The mesentery is found congested & contains multiple, soft LNs

3- Appendectomy is performed & the post-operative course is usually uneventful

Tuberculous mesenteric adenitis (tabes mesenterica)

Definition

This is a childhood type of intestinal TB that is comparable to the 1st complex of the lung but is less common.

Etiology

- Organism: Mycobacterium tuberculosis
- Route: ingestion of contaminated milk.

Clinical picture

During activity

Symptoms

General: TB toxæmia, low grade fever, sweating, wasting & pallor.

Local: Abdominal pain with vomiting & diarrhoea.

Signs

1- In acute cases → severe tenderness, rebound tenderness & rigidity in the right iliac fossa (simulating appendicitis) or along the line of the mesentery.

2- Firm irregular tender masses may be felt (nodes).

Investigations

A-Laboratory

- 1- Leucocyte count is normal.
- 2- Tuberculin test: If the test is negative → TB is excluded.
- 3- PCR.

B-Imaging

X-ray of the abdomen

May show calcified nodes as mottled opacities arranged along the line of the small intestinal mesentery.

Treatment

- For active cases → Antituberculous.

-Pathology:

It consists of:

1- A small focus in distal ileum (**Peyer's patches**).

2- Huge enlargement of mesenteric nodes.

Fate: The nodes may undergo either →

a) Healing by fibrosis and calcification

b) Caseation occurs → a mesenteric cold abscess

- Calcified uncomplicated nodes need no treatment.

Retroperitoneal tumours

A-Renal and adrenal tumours

Discussed in urinary tract tumors chapter

B- Retroperitoneal sarcoma

Clinical picture

Symptoms

- 1- The patient may complain of vague abdominal pain.
- 2- If lesion infiltrates the colon → chronic large bowel obstruction.
- 3- If the ureter is obstructed → renal pain.

Signs

- 1- Fixed & hard abdominal mass

The retroperitoneal space

- is that portion of the body which is bounded by:

Anteriorly: Posterior peritoneum.

Posteriorly: the spine & posterior abdominal wall

Muscles

Superiorly: the 12th rib & attachments of the diaphragm

inferiorly: extends to the pelvis.

- There are no anatomic barriers in this area → pathological processes extend easily through it.

(The size of the lesion outmatches its symptoms)

Investigations

Ultrasound and CT scan

- are very useful for the diagnosis.
- CT scan accurately localizes the lesion, its size and reveals infiltration of adjacent structures



Treatment

- 1-The patient is explored for the possibility of resection.
- 2-Unfortunately, most of the cases are unresectable and all what can be done is to take a biopsy.
- 3-Radiotherapy may offer some palliation.

C- Retroperitoneal lipoma.

- More common in females
- it grows slowly and may attain a large size.
- It is prone to malignant transformation.

D- Retroperitoneal lymphoma.

Acute Appendicitis

Pathology

Gross Types :

1) obstructive acute appendicitis :

- In $\frac{2}{3}$, inflammation is initiated by obstruction of the lumen
- Obstruction is usually caused by:
 - a) A faecolith (inspissated stool)
 - b) Swelling of the lymphoid tissue in response to viral infection.
 - c) Kinking by adhesions
 - d) Intestinal parasites as pin worms
 - e) Tumours of the appendix or the caecum

less common

-Distention of the obstructed appendix → overgrowth of the resident bacterial flora → invasion of the mucosa → rapid Suppurative or gangrenous appendicitis

2) Non-obstructive acute appendicitis (in $\frac{1}{3}$ of cases)

-Inflammation usually starts in the mucosa and tends to remain there (catarrhal inflammation)

-may spread but at slow rate and unlikely to perforate

Course

1-Acute appendicitis may resolve but liable for recurrence

2-Persistent obstruction (with a faecolith) → gangrene & perforation → generalized or localized peritonitis (depending on the severity of inflammation & efficiency of the body defense)

a) When the inflammatory process is slower → the body defense has the time to surround the inflamed appendix by adhesions to nearby intestine and the omentum → appendicular mass (phlegmon) within 3 to 5 days of the start of pain in the right iliac fossa or in the pelvis.

b) Perforation of the appendix within an appendicular mass → an appendicular abscess.

3- Portal pyaemia (pylephlebitis) "rare"

-results from Suppurative thrombophlebitis of the portal venous system

-present with : Chills, high fever, low-grade jaundice and hepatic abscesses (the hallmarks)

Clinical picture

Diagnosis is mainly clinical

Symptoms

1- Migrating right iliac fossa pain (presenting symptom)

a) **starts** in the periumbilical region & ill localized (visceral pain)

-is due to distention of the appendix

-Felt in this area because the appendix is a part of the midgut.

b) **pain shifts** to the right iliac fossa Within 6-10 hours & becomes sharper and more localized (somatic pain).

- is due to irritation of the parietal peritoneum

-The pain is aggravated by movement or cough.

Prevalence

- The **most common** acute abdominal surgical emergency.

• Age:

a) Most prevalent in 2nd and 3rd decades of life after which risk decrease with age.

b) Rare in infant as appendix is wide-mouthed and well drained

c) Rare in elderly as the lumen is almost obliterated and the wall contains little lymphoid tissue

• Race: more common in Western countries and in urban than rural areas due to low fibre diet



Sites of appendicular perforation :

a) at the tip (where the appendicular vessels are very close to wall)or

b) intramural

c) at the site of impaction of the faecolith

- The incidence of perforation is high in the very young < 5 years and in the elderly.

- In untreated cases the course of the disease is variable

2- Anorexia and nausea are nearly always present.

3- Vomiting occurs in about $\frac{3}{4}$ of the patients

-is not usually repeated, but is always preceded by the pain

4- Constipation is usual.

Signs (affected by stage of the disease & position of appendix)

1. Temperature and pulse : slightly elevated early in the disease.

-Higher elevations indicate a complication, such as perforation.

2. Cough tenderness: If the patient is asked to cough, the pain becomes sharp and well localized to the site of the appendix. He is then asked to point to the area of maximum pain.

3. Localized tenderness & rebound tenderness → usually in the right iliac fossa over the McBurney's point (is at the junction of inner $\frac{2}{3}$ & outer $\frac{1}{3}$ of a line extending from the umbilicus to the ASIS)

4. Rigidity → indicates perforation.

5. Restricted abdominal wall movement with respiration → indicates peritonitis.

6. a tender appendicular mass may be felt in the right iliac fossa if the patient presents on the 3rd or 4th day of the attack.

7. if Rigidity is present and the size of the mass increases →

Appendicular abscess is formed (means accumulation of pus inside an appendicular mass)

- is suspected by persistent pain, rising pulse rate and higher temperature with deterioration of the general and local conditions

8. Pelvic examination (vaginal or Rectal)

- Reveals tenderness or a mass in case of pelvic appendicitis.

- To exclude gynecological problems.

9. Special signs (assist in the diagnosis if present)

a. **Rovsing's sign**: is pain felt in the right lower quadrant on deep palpation of the left lower quadrant.

b. **Psoas sign**: is pain on extension of the right thigh.

- Cause → psoas spasm due to irritation by retrocaecal appendicitis.

c. **Obturator sign**: is pain on internal rotation of the flexed RT thigh

- caused by irritation of the obturator internus by pelvic appendicitis

Atypical clinical features

1- Tenesmus (from rectal irritation) and dysuria (from bladder irritation) if Inflamed appendix points deeply in the pelvis
- Abdominal signs are minimal and tenderness is elicited on pelvic examination.

2. Pain and tenderness are higher than the McBurney's point when the caecum is high.

3. pain felt in the right loin in a long retrocaecal appendix

-Abdominal examination reveals minimal tenderness or guarding.

4. Diarrhea due to ileum irritation by an inflamed retro-ileal appendix.

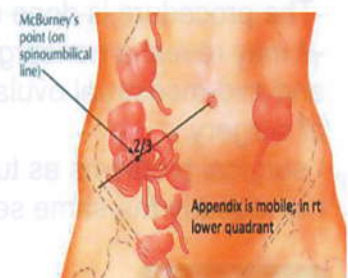
5. Appendicitis in infants and young children < 3 years is more serious→ 80% of patients will have perforated appendicitis due to:

a. Difficulty of examination of an infant or young child.

b. The greater omentum is not well developed so no localization

c. prominent vomiting → wrongly diagnosed as gastritis or enteritis

- Proper history taking & physical examination are the corner stone of diagnosis
-If vomiting precedes the abdominal pain, first consider gastroenteritis
-if the patient feels hungry and can eat → uncertain to be acute appendicitis
-site of localized tenderness may be low in pelvis or high in RT hypochondrium according to site of appendix
- Appendicitis does not start by a rigor or a temperature above 40°C.
-Subhepatic appendix is commonly misdiagnosed as cholecystitis



6. Acute appendicitis in the elderly is dangerous as it:

- a) Produces little tenderness and rigidity → perforation is common
- Eliciting these signs is even more difficult in obese patient
- b) The immune system weakens with age

7. Appendicitis with pregnancy.

- a) The site of pain is displaced upwards as pregnancy progresses.
- b) Difficult omental localization and is misdiagnosed as pyelitis.
- c) If the diagnosis and operation are delayed until perforation, there is a higher chance of abortion or premature labor (**50%** of cases)

Investigations

1. Leucocyte count.

- A moderate leucocytosis (10,000-16,000/uL) with a predominance of neutrophils in **80-90%** of cases.

2. Urine analysis may show few red blood cells.

- The presence of a large number of RBCs or pus cells raises the suspicion of a ureteric calculus or urinary tract infection respectively

3. Pregnancy test if ectopic pregnancy is suspected.

4. Ultrasound examination

- is easy, inexpensive and can be used in pregnant patients.
- In acute appendicitis: a) the appendix is > 6 mm in diameter
- b) the wall is thickened c) there is periappendiceal fluid
- d) the appendix is not compressible e) faecolith may be detected.
- detect appendicular abscess if present
- exclude other diseases that simulate acute appendicitis as:
 - a) Gynecological problems in females → do transvaginal US
 - b) Suspected right ureteric stones → back pressure changes will be shown in right kidney.

5. CT scan will demonstrate: a) dilated appendix more than 6 mm, thickened wall and thick mesoappendix.

b) identify other inflammations mimicking acute appendicitis.

6. Laparoscopy.

- is the policy in some centres to perform laparoscopy for females in the child-bearing period presenting with lower right abdominal pain.
- The procedure is done under general anaesthesia.
- It may reveal a nonsurgical problem as pelvic inflammatory disease and mid-menstrual ovulation pain → the patient is spared the trouble of surgery.
- surgical problems as tubal pregnancy & acute appendicitis can be managed in the same session by open or laparoscopic surgery.

Treatment

I- **Uncomplicated acute appendicitis**

- Treatment is by urgent appendicectomy
- The operation should not be delayed especially in children, elderly persons, pregnant females and diabetic patients.

Laparoscopic Appendicectomy

main advantage is:

- a) its exploratory potential (whole peritoneal cavity)
- b) Less morbidity
- c) less pain
- d) less hospital stay
- e) faster recovery

- low leucocytic count does not exclude acute appendicitis but high count with shift to the left supports the diagnosis

- If at operation the appendix looks normal :

- 1) The surgeon should check :
 - a) terminal ileum (Meckel's diverticulum , mesenteric adenitis and crohn's disease
 - b) right fallopian tube & ovary
- 2) up to **20 %** -ve appendicectomy is acceptable (better than to miss acute appendicitis that proceeds to rupture)

- Some surgeons nowadays oppose doing interval appendicectomy after appendicular abscess

II- Appendicular mass

-Urgent appendicectomy is not recommended because:

1. An appendicular mass represents success of the body to isolate the danger → better left undisturbed.
 2. Appendicectomy is technically difficult and carries the hazard of injuring the adherent intestine leading to peritonitis or faecal fistula.
- Initial conservative treatment is done named **Ochsner-Sherren**

III- Appendicular Abscess

- Nowadays, ultrasound guided aspiration of pus is usually performed leaving a pigtail catheter for drainage.
- If ultrasound aspiration fails , open surgery is performed→ all loculi are opened, the pus drained and a rubber drain is left in place for a few days .
- we do not remove the adherent friable appendix at this stage.
- A pelvic abscess , is drained transanally through the anterior wall of the rectum.
- The patient is discharged within days and is scheduled for "interval appendicectomy" after **3-6 months**

IV-Perforated appendicitis with generalized peritonitis

- Urgent surgery is required.
- The perforated appendix should be removed and peritoneal toilet is performed→ performed via either laparoscopy or laparotomy

Differential Diagnosis of acute appendicitis

Group 1: Thoracic problems

Right sided pneumonia or right basal pleurisy

- abdomen is not rigid with minimal tenderness and marked chest symptoms

Group 2 : Upper abdominal problems

1. Perforated peptic ulcer

- history of chronic dyspepsia.

-When perforation occurs there is a sudden onset of severe upper abdominal pain , tenderness and mild shock followed by steadily increasing peritonitis stage that present with → gradually increasing pain, tenderness and progressing rigidity.

-The rigidity may be very marked in the right iliac fossa because gastric or duodenal contents flow along the right paracolic gutter to the right iliac fossa.

- However, tenderness and rigidity are more marked in the right hypochondrium and epigastric regions.

- A plain X-ray usually shows gas under the diaphragm.

2. Acute cholecystitis.

- the signs and symptoms may resemble appendicitis but they are definitely more marked in the right hypochondrium rather than the right iliac fossa.

-Ultrasonography will confirm the diagnosis.

3. Cyclic vomiting of children.

- temperature is normal, rigidity never occurs and the leucocytic count is normal.

Ochsner-Sherren

regimen includes:

1. Rest in bed for the first few days.
 2. IV fluids are administered until nausea subsides and the patient is able to start oral feeding with fluids followed by gradual introduction of solid meals.
 3. Antibiotics: A combination of ampicillin, aminoglycoside and metronidazole administered IV at first then orally (cover most of the pathogenic organisms)
 4. An analgesic may be prescribed for the pain.
 5. Careful observation of the temperature, pulse, pain, vomiting, degree of tenderness and rigidity and for the size of the mass.
- **In 80-90%** of patients the appendicular mass will resolve under conservative treatment.
- The patient is discharged and is scheduled after 3 months for interval appendicectomy, If the operation is not performed → risk of a further attack of acute appendicitis

Group 3. Lower abdominal problems

1. Non specific mesenteric lymphadenitis.
 - The commonest organism is Yersinia.
 - The patient is usually a child with pain which comes in attacks.
 - The tenderness shifts with changing of position.
 - It is difficult to be sure about the diagnosis.
2. Iliac adenitis.
 - a demonstrable cause for the adenitis and the site of maximum tenderness is much lower near the iliac vessels.
3. Cancer of the caecum.
 - This is more likely to be misdiagnosed as appendicular mass.
4. Regional ileitis.
 - In its acute form it may be indistinguishable from appendicitis unless a vague doughing mass can be felt.
 - A definite history of intestinal trouble with repeated diarrhea and bleeding is very suggestive.
5. Meckel's diverticulitis.
 - It is usually not diagnosed except after exploration.
6. Gastroenteritis.

Group 4: Pelvic problems

1. Mid-cycle ovulation pain (Mittelschmerz) due to ruptured graffian follicle
 2. Ruptured ectopic pregnancy.
 - There usually pallor, tachycardia and hypotension.
 3. Tubal abortion.
 4. Twisted ovarian cyst.
 5. Pelvic inflammatory disease (PID), e.g. salpingitis, pyosalpinx & tubo-ovarian abscess.
 - There usually pyrexia.
 6. Red degeneration in a fibroid.
- Sonography is very helpful in the diagnosis of these problems

Group 5 : Urological problems

1. Right renal ache or ureteric colic (right ureteric stone).
2. Right pyelonephritis.
 - Often start with a rigor and temperature 39-40°C.
 - Pain is more in the loin rather than iliac fossa.
 - Pain radiates to the upper thigh, testicle and suprapubic area.
 - Often associated with dysuria and/or pyuria

Group 6: Neurological problems

- The pain which precedes the eruption of herpes zoster affecting 10th, 11th and 12th thoracic nerves.
- The pain is very similar in site and distribution to appendicitis but there is no tenderness or rigidity and the pain does not shift.
- Eruptions may appear 36-48 hours later.

Common differential diagnoses of

Acute appendicitis:

- a) Right tubo-ovarian causes
- b) Mesenteric adenitis (Yersinia).
- c) Right ureteric stone
- d) Gastroenteritis
- e) Meckel's diverticulitis

- Problems of the female genital system may resemble appendicitis to a great extent

- A mass in the RT iliac fossa is most likely appendicular mass

- DD of an appendicular mass

1.Carcinoma of the caecum

The condition is a chronic & is likely to be associated with anaemia & weakness.

- The mass usually hard & not tender

2.Crohn's disease

3. Hyperplastic ileocaecal tuberculosis.

4. Right iliac lymphadenitis

Neoplasms of the Appendix

I-Carcinoid Tumour (Argentaffinoma)

Incidence

This is the commonest tumour of the appendix.

- carcinoid tumour affects the gut at :
 - a) appendix in **65%** of cases (commonest site)
 - b) The ileum in **25%**.

Pathology

- Origin: from Kulchitsky cells in the depth of the mucosal pits.
- Course: usually benign but a minority (5%) invade & metastasize to the liver.
- Examination of the cut section shows that the tumour appears to arise from the muscle layer and has a characteristic bright yellow color caused by its lipid content
- The distinction between benign and malignant tumours depends mainly on the biological behaviour:
 - a) Tumors <1 cm are usually benign
 - b) Tumors >2cm are almost always malignant and metastasize which may produce the rare carcinoid syndrome because of their production of serotonin

Clinical picture

- 1- Commonly asymptomatic.
1. Carcinoid tumour is sometimes an accidental histological diagnosis in an appendix that has been removed because of chronic or acute appendicitis.
2. Carcinoid syndrome is rare and is produced only by malignant carcinoid tumours that metastasize to the liver , it includes one or more of the following:
 - a) flushing with attacks of cyanosis
 - b) bronchospasm
 - c) diarrhea with borborygmi
 - d) pulmonary stenosis

Treatment

- 1- appendicectomy if tumour <2 cm and confined to appendix
- 2- right hemicolectomy for larger lesions ≥ 2 cm

II-Adenocarcinoma

- Incidence: is rare.
- It may present as acute appendicitis.
- behaves as carcinoma of the colon.
- Treatment: is right hemicolectomy If it is confined to the appendix and the regional lymph nodes

Chronic Appendicitis

(More correctly recurrent subacute appendicitis)
- present with recurrent attacks of abdominal pain and dyspepsia.
- In most of the cases it is difficult to prove that chronic appendicitis is the cause of pain. Even if pain and tenderness are centered around the right lower abdomen
- it is advisable to exclude other causes of chronic pain before removing the appendix as : Amoebic colitis, irritable bowel syndrome, chronic calcular cholecystitis, and Crohn's disease
- treatment is appendicectomy (Preferred by laparoscope)

- The diagnosis of the carcinoid syndrome is confirmed by finding high urinary concentrations of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA).

Composition

The main constituents of bile are:

1. Water.
2. Electrolytes → same concentration as the plasma.
3. Bile salts.
 - The major bile acids which form these salts are :
 - a) Cholic b) Deoxycholic c) Chenodeoxycholic acids.
 - These acids conjugate with taurine or glycine to form bile salts
4. Cholesterol and phospholipids.
 - Cholesterol is insoluble in water and is carried in bile by the combined detergent effect of bile salts and phospholipids.
5. Bile pigments.
 - Bilirubin diglucuronide is the break down product of haemoglobin.
 - is responsible for the yellow color of bile

Gallbladder Function

1. Stores and concentrates bile.
 - Sodium, chloride, and water are selectively absorbed → 10-fold increase in concentration of bile salts, bile pigments & cholesterol.
2. Mucus secretion
 - protects the mucosa from the lytic action of bile and facilitates passage of bile through the cystic duct.
3. Contraction of gall bladder and empties bile for digestion.

I-humoral stimulation:

- Food → release Cholecystokinin from intestinal mucosa →
 - a) Contraction of the gall bladder
 - b) Relaxation of the sphincter of Oddi.
- } evacuation of 70% of GB within 30 min.

II- nervous stimulation

- Vagal innervations → stimulates contraction
- Sympathetic stimulation → inhibits motor activity

Bile

-Amount: Normal adults secrete 250-1000 ml bile per day

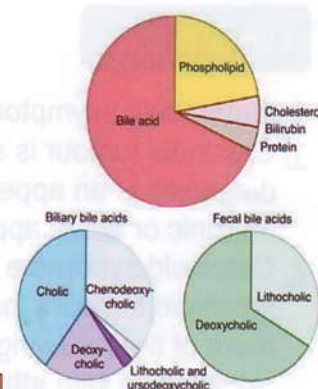
-Control:

1-Neurogenic:

- a) Vagal stimulation → increases secretion
- b) splanchnic stimulation → decreases bile flow

2- Hormonal

- Hydrochloric acid, protein breakdown products and fatty acids → stimulate secretin release from the duodenum → stimulate bile secretion



Imaging investigations

I- Abdominal ultrasound

-The 1st investigation performed in most biliary system disorders

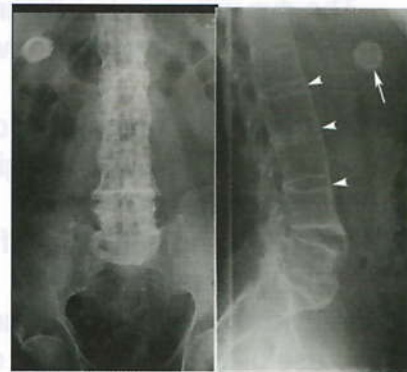
- Advantages:

- a) It is easy, simple, inexpensive and non invasive investigation.
- b) Done in the presence of acute inflammation or jaundice.
- c) It can provide the following information:
 - 1- Detection of gall bladder stones in a **98%** of cases
 - 1- detection of stones of CBD (**less accurate**)
 - 2- Detection of the thickness of the wall of the gall bladder.
 - 3- Visualization of dilated extra and intrahepatic biliary ducts in patients with surgical obstructive jaundice.
 - 4- Detection of masses in the porta hepatis or head of pancreas



II- Plain x-ray (of a limited value) detects :

- Gall stones in only **10-15%** of cases
- A gall bladder with a calcified wall (porcelain) can be seen.
- Air in the gall bladder (in acute emphysematous cholecystitis)
- Air in the biliary system (after choledochoduodenostomy or sphincterotomy operations)



III- Oral cholecystography:

- was previously the investigation of choice for detection of gall stones but now replaced by ultrasonography

IV- Endoscopic retrograde cholangiopancreatography (ERCP)

Method

- An endoscope passed through the oesophagus, stomach & duodenum
- The duodenal papilla is localized and a cannula is passed in it→ a contrast material is injected →visualize the common bile and the pancreatic ducts by radiography.

Indications:

- 1- Diagnosis of lesions involving the lower end of CBD as carcinoma of head of pancreas or ampulla of Vater (a biopsy can be taken)
- 2- Detection of missed calculi in the CBD after cholecystectomy.
- 3- Detection of operative injuries of the biliary system.
- 4- Visualization of the pancreatic duct in patients with chronic pancreatitis or pancreatic pseudocyst.
- 5- Therapeutic procedures can be performed with ERCP:
 - a) Sphincterotomy and removal of bile duct stones.
 - b) Insertion of a stent to drain malignant obstruction of the duct



Complications

- 1) Ascending cholangitis (**most common**) or septicaemia after injection of the contrast material in presence of unrelieved obstruction in the CBD due to increased pressure in of the CBD
- 2) Acute pancreatitis or duodenal perforation



V- Magnetic resonance cholangiopancreatography (MRCP)

- provide good quality images without the complications associated with ERCP or PTC
- diagnostic only not therapeutic

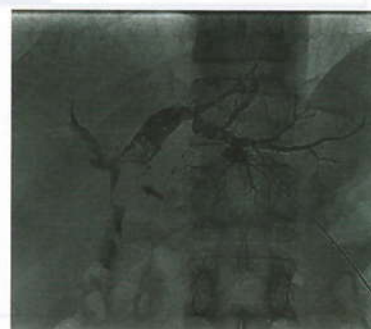
VI- Percutaneous transhepatic cholangiography (PTC)

Pre-requisites

1. Normal coagulation.
 - The prothrombin time (PT) and concentration are checked.
 - If PT is prolonged → vitamin K is given I.V for a few days to correct hypoprothrombinaemia before the procedure.
2. Dilated intrahepatic biliary radicles (seen on ultrasound)

Technique

- Under local anaesthesia, a thin cannula (**Chiba needle**) is introduced through the **8th** intercostal space into the liver and



continuous suction by a syringe is applied until bile is aspirated and the dye is then injected.

- The dye will visualize the intrahepatic biliary ducts and then view the bile duct from above downwards

Indications

Diagnose high obstruction of bile ducts as in post-operative biliary strictures or in hilar cholangiocarcinoma.

Complications

- 1) Bleeding may occur if there is hypoprothrombinaemia.
- 2) Biliary peritonitis

VI- Endoscopic ultrasound

- useful for the diagnosis of ampullary or pancreatic carcinoma

VII- Radioisotope scanning (HIDA scan)

Method

- ^{99m}Tc labeled derivative of iminodiacetic acid (HIDA) is injected IV $\rightarrow$ rapidly excreted by the liver to visualize the biliary tree.

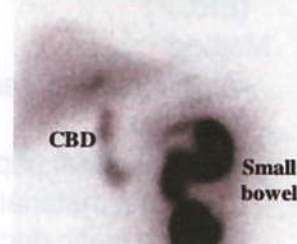
Uses

- 1) Diagnosis of acute cholecystitis.
 - Non-visualization of the gall bladder = obstruction of its neck
 - A gall bladder that fills by the isotope excludes it
- 2) Diagnosis of congenital biliary atresia.
- 3) To visualize a biliary enteric anastomosis

Normal HIDA scan



Acute cholecystitis



Gall stones

Incidence

- More common in Western countries (due to dietary factors), occur in at least 20% of women over the age of 40 in these countries.
- A female: male ratio of 3: 1 in cholesterol and mixed stones.
- increased females incidence is not present after the age of 60 and with pigment stones.
- The incidence of gallstones increases with age.
- There is familial incidence of the disease (more common in parents and siblings of patients)

Definitions

Cholelithiasis

Gall bladder stones

Choledocholithiasis

Common bile duct stones

Cholecystitis

Inflammation of gallbladder

Cholangitis

Infection of bile ducts

Types and composition

	Pure cholesterol stones	Mixed stones	Pigment stones	
			black	Brown
Incidence		90%	in patients with haemolytic anaemias or cirrhosis.	-infected stagnant bile(in the bile duct) -in the presence of foreign bodies as biliary stent

Constituents	100% cholesterol	Cholesterol (> 60%), calcium bilirubinate, calcium phosphate & calcium palmitate	calcium bilirubinate	calcium bilirubinate (mainly), calcium palmitate and cholesterol
Number	Single (Sometimes multiple)	multiple	multiple	multiple
Size	>2.5 cm	0.5-2.5 cm	< 2.5 cm.	< 2.5 cm
Shape	Rounded	faceted	multiple spicules	laminated
Color	Yellow	yellowish	tarry black	brown
x-ray	radiolucent	radio-opaque in 15 %	Radio-opaque in 50%	Radio-opaque



Etiology

I-Cholesterol and mixed gall stones

A) Disturbed bile salts cholesterol ratio.

- Cholesterol is insoluble in water
- carried in bile by the combined detergent effect of bile salts and phospholipids which form clusters
- A certain ratio (25: 1) between bile salts & phospholipids on one hand and cholesterol on the other hand has to be maintained to keep cholesterol in solution
- Any lowering of this ratio → supersaturated bile (**lithogenic bile**) → cholesterol precipitation.
- The following factors disturb this ratio:
 - 1) Reduced bile salt pool.
 - Malabsorption of bile salts in the terminal ileum as in:
 - i) Crohn's disease
 - ii) small bowel resection
 - iii) bypass procedures.
 - In DM autonomic neuropathy of the ileum may affect the enterohepatic circulation & the bile acid pool.
 - Diminished hepatic synthesis in liver disease.
 - Estrogens reduce the concentration of bile salts in bile.
 - 2) Increased cholesterol synthesis.
 - occur in obesity, high dietary fat and high caloric diet.

Liver cirrhosis is associated with an increased incidence of gallstones overall, but black pigmented stones are commonest

-Most gallstones are silent and remain so in 70% of patients, being diagnosed as an incidental finding on ultrasound.

.Symptoms (recurrent biliary colic) develop in 2-3% per year.
.Each year, 3-5% of symptomatic patients develop complications

B) Stasis of bile:

- 1) Female hormones.
 - Progesterone → relax and impair emptying of the gall bladder.
 - Estrogen, oral contraceptives and repeated pregnancy → increased incidence of gallstones.
- 2) Following truncal vagotomy due to denervation of the gall bladder.
- 3) Diabetes mellitus.
- 4) Obesity.
- 5) Long term parenteral nutrition
lack of oral intake → prevent release of cholecystokinin.

II- Pigment Stones

A) black pigmented stones

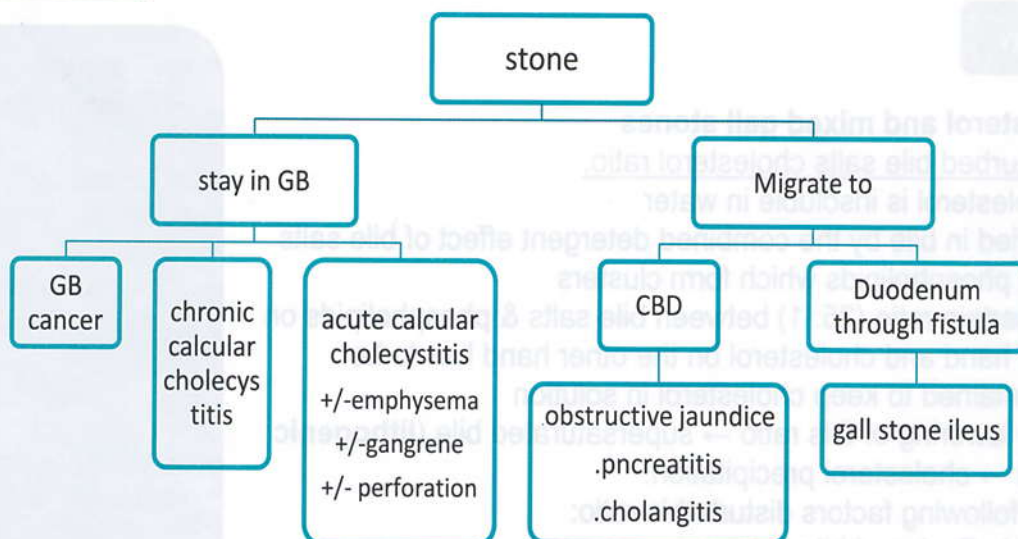
- 1- Haemolytic anaemias.
Any condition that shortens the life span of RBCs as: hereditary spherocytosis, thalassaemia or prosthetic heart valves
- 2- Liver cirrhosis due to decreased secretion of bile acids by the cirrhotic liver → diminished solubility of unconjugated bilirubin.

B) Brown pigment

Infection :

-by some strains of E. coli → production of B- glucuronidase enzyme → hydrolyses bilirubin glucuronide into the insoluble bilirubin which precipitates as calcium bilirubinate.

Complications



A) Complications in the gallbladder

- 1- Obstruction of the cystic duct or neck of gall bladder :
 - i) Mucocoele of the gall bladder (if the contents remain sterile)
 - ii) Acute calcular cholecystitis (If contents infected)with or without empyema, gangrene or perforation.
- 2- Chronic calcular cholecystitis.
The gall bladder is usually thick-walled and contracted
- 3- Carcinoma of the gall bladder.
 - long-standing calcular disease → squamous metaplasia → carcinoma.
 - About 90% of patients with carcinoma of the gall bladder have gallstones.

B) Complications caused by migration of stones

I- Migration to CBO

1- Obstructive jaundice

-Lead to bleeding tendency, septicaemia, renal & hepatic failure.

2- Cholangitis and cholangitic abscesses of the liver.

3- Acute pancreatitis :

- If a stone in the lower end obstructs the common channel of CBD and pancreatic duct.

-in many cases of calcular pancreatitis, there is no actual obstruction of the ampulla by a stone during the attack , it is suggested that the passage of a stone through the ampulla may precipitates pancreatitis (an unknown mechanism)

4- Biliary cirrhosis: rare in long-standing intermittent obstruction

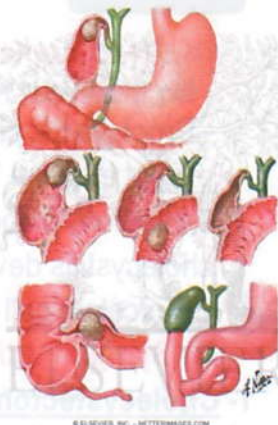
II-Migration to duodenum through a fistula

• Gallstone ileus (rare)

-A big stone (> 2.5 cm) passes through a fistula between the gall bladder and duodenum → impacted in the terminal ileum (about two feet from the ileocaecal valve) → intestinal obstruction (called gallstone ileus obstruction)

- Clinical diagnosis is usually difficult as the patient is often an elderly and gives a long history of biliary problems.

-The symptoms of intestinal obstruction are usually misinterpreted as being due to biliary problems



Clinical picture

A) **Asymptomatic** (silent) → **most common**

- Accidentally discovered by plain X-ray or ultrasound performed for another abdominal problem

B) **Symptomatic** :

1- **Recurrent attacks of biliary pain.**

-Incidence: The usual complaint

-Site : right hypochondrium

-referral : to the back of the right chest or right shoulder.

-Course : constant, increases in severity over 30 minutes

-Duration : lasts < 5-6 hours (longer = acute cholecystitis)

-precipitated by: intake of a fatty meal.

-associated symptoms : nausea or vomiting during the attack.

2- **Biliary dyspepsia.**

- Fat dyspepsia with bloating and excessive eructations following fatty meals.

- Sometimes, these patients are diagnosed as having peptic ulcer or chronic colitis.

3-**Reflex symptoms.**

- Reflex retrosternal pain → usually diagnosed as anginal pain (cholecystocardiac link).

- Actual ECG changes may occur

Signs

1- tenderness in RT hypochondrium.

2-Murphy's sign positive:

-the gall bladder is palpated while the patient is asked to take a deep breath → the patient will catch her breath

-**Complications**
(may be the 1st presentation)
-Obstructive jaundice

Investigations

Abdominal ultrasound (investigation of choice)

- simple , inexpensive and non-invasive technique
- It can provide the following information:
 - 1- Detection of gall stones (in **98%** of cases)
 - 2- Thickness of the gall bladder wall → thickened wall means long-standing recurrent inflammations
 - 3- The diameter of the CBD and any intrahepatic biliary dilatation (if present = a stone in the CBD)

Ultrasound

- can see almost all gall bladder stones (98%)
- only 1/3 of CBD stone

Treatment

A) Asymptomatic cases :

- No treatment needed
- Prophylactic cholecystectomy not indicated for silent stones except in some 70% of people over 20 years as:
 - 1-in elderly diabetics (increased morbidity and mortality if acute cholecystitis develops).
 - 2-Porcelain gall bladder (not considered nowadays precancerous)

B) Symptomatic :

1- Cholecystectomy (The **standard** treatment)

- done either by the open or laparoscopic method
- 2- Extracorporeal shock wave lithotripsy (ESWL) & medical dissolution of gallstones
 - had some popularity in the 1980s.
 - not used now because of limited efficacy and many side effects



Acute calcular cholecystitis

Pathology

Gross picture

- The gall bladder appears:

Shape: distended

Wall: thickened and show multiple micro-abscesses

Serosa: loses its bluish luster & covered by fibrinous deposits.

- As a defensive mechanism → the greater omentum, the duodenum and colon become adherent to the gall bladder to localize infection.

Consequences

1- Resolution (most cases)

- with treatment, the stone dislodges and the obstruction is relieved with gradual resolution of inflammation.

2- obstruction persists (less common)

Lead to progressive distension → thrombosis of the blood vessels

→ gangrene → perforation → peritonitis either:

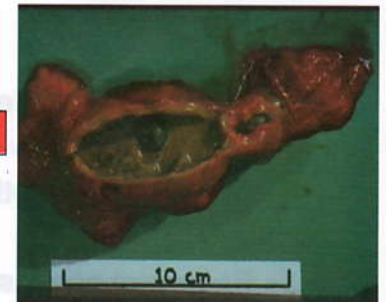
A) localized

B) Generalized (0.5%).

B) Empyema of gall bladder.

The gallbladder is converted into a closed bag of pus.

C) Recurrent attacks → lead to chronic calcular cholecystitis



-Etiology

Calcular (**98%**) :

2nd to obstruction of the cystic duct or Hartmann's pouch by a stone

-Pathogenesis

Obstruction of the cystic duct or neck of GB by a stone

→ infection "by Gram -VE bacilli

"→ acute cholecystitis

Clinical picture

Symptoms

1- Abdominal pain :

- At first : diffuse colicky upper abdominal pain
- later : with inflammation of the serosa → dull aching pain, persistent and localizes in the right hypochondrial region.
- referred to : the right shoulder (due to irritation of the diaphragm by sensory fibres of the Phrenic nerve)
- duration : pain > 6 hours = acute cholecystitis.

2- Nausea or vomiting

Signs

- a) General: 1- pyrexia 2- tachycardia
 3- Jaundice (**Mirrzi syndrome**)

b) Local :

- 1- Abdominal examination reveals tenderness, rebound tenderness and muscle guarding in the right hypochondrium.
- 2- difficult to palpate the distended gall bladder due to the overlying tenderness and rigidity.

3- Special signs :

i- Murphy's sign

If pressure is applied to the right hypochondrium and the patient is asked to take a deep breath → he will hold his breath.

ii- **Boas's sign**

area of hyperesthesia between the 9th -11th ribs on RT posterior

Investigations

Laboratory

- 1- Blood picture.
Polymorph nuclear leucocytosis may be present.
- 2- Liver function tests : usually normal.

Imaging

Abdominal ultrasound (the most appropriate)

- reveal :a) Presence of gallstones
- b) Distended gall bladder with thickened wall showing microabscesses and serosal edema due to spreading inflammation.

Treatment

I-Early cholecystectomy (within 3 days)

- prerequisites: 1) diagnosis is confident
- 2)the patient is fit
- 3) the surgeon is experienced
- has the following advantages :
 - 1- Early surgery is not difficult as adhesions are fibrinous not fibrous
 - 2- avoids the complications of acute cholecystitis that may arise during conservative treatment.
 - 3- One hospital admission with early return to work.

Acute
emphysematous
cholecystitis"

- A special form may occur in diabetic pts. due to infection by anaerobic organisms, e.g. clostridia.
- is highly virulent & accompanied by gas formation → early gangrene of the gall bladder

- Mirrizi syndrome

A large stone impacted in the Hartmann's pouch → compress the bile duct from outside → jaundice
- Pain persisting more than 6 hours denotes acute cholecystitis

Differential Diagnosis

1. Perforated duodenal ulcer
2. Acute pancreatitis.
3. High retrocaecal appendicitis.
4. Right pyelonephritis.
5. Amoebic hepatitis

The definitive treatment is cholecystectomy

II- Initial conservative treatment followed by cholecystectomy after 6 weeks

- 1- Nil by mouth and IV fluids.
 - 2- Antibiotics: against gram negative bacilli as Ampicillin or cephalosporin.
 - 3- Analgesics : as pethidine or NSAIDs.
 - 4- Follow-up of the pulse, temperature, area of tenderness & rigidity.
- Most patients improve under this treatment
 - The patient is sent home and is advised to come back for elective cholecystectomy after 6 weeks to allow the adhesions to subside.
 - If the patient deteriorates during ttt → surgery should be performed
- a) Cholecystectomy if safely feasible.
 - b) Cholecystostomy if dense adhesions present which makes cholecystectomy a risky operation.

Cholecystostomy

- The gall bladder fundus is opened and the stones are removed.
- The gall bladder is drained by a tube for one week
- The patient usually returns for elective cholecystectomy after 6 weeks.

Acute non-calicular cholecystitis

Etiology

- a rare and serious condition which may occur in:
 - 1- Patients suffering from major burns or major trauma.
 - 2- Patients on prolonged total parenteral nutrition.
 - 3- It may complicate certain infections as brucellosis and typhoid.
- The actual cause and pathogenesis are unclear, may be due to Change in the composition of bile or ischemia of the gall bladder

Clinical picture

- is similar to that of acute calculus cholecystitis, but usually the diagnosis is delayed because it is not suspected

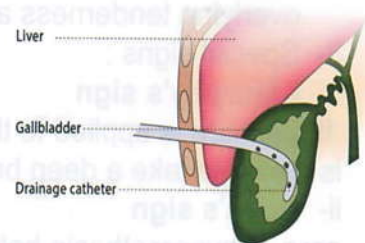
Investigation

Ultrasound examination is diagnostic.

Treatment

Urgent cholecystectomy

Cholecystostomy (drainage)



Jaundice

Definition

- Yellowish discoloration of the body tissues and fluids (except the brain, CSF, tears, saliva and milk) which results from accumulation of bilirubin in blood.
- It becomes manifest when serum bilirubin exceeds 2.5 mg/dL
- Normal bilirubin level 0.2-0.7 mg%

-Jaundice should be looked for in day light

- In people with dark skin the sclera is normally yellowish so look for yellow coloration in the mucous membrane of the mouth.

Bilirubin metabolism

1- RBCs destruction.

- The reticuloendothelial cells particularly in the bone marrow and the spleen phagocytose old RBCs

2. Breakdown of haemoglobin into :

- Globin → enters into the amino acid pool
- Heme → transformed into →
 - Iron
 - Biliverdin → reduced to bilirubin (water insoluble → transported in plasma bound to albumin → unconjugated bilirubin)

3. Bilirubin conjugation.

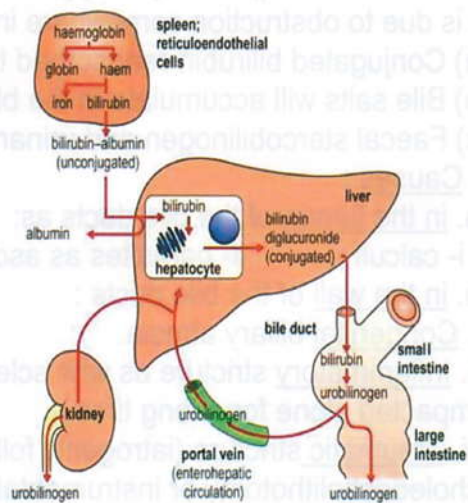
- Site: in the liver
- Bilirubin + glucuronic acid → bilirubin diglucuronide (conjugated bilirubin which is water soluble).
- Is catalyzed by the bilirubin glucuronyl transferase enzyme.

4. Bilirubin excretion.

- Conjugated bilirubin is excreted via the biliary passages to the intestine.

5. Changes in intestine and reabsorption .

- Site of reabsorption: In the terminal ileum and colon
- Bacterial enzymes convert conjugated bilirubin to stercobilinogen → oxidized to stercobilin (give the normal color of stools)
- Up to **20%** of stercobilinogen is reabsorbed → 90% of this is reexcreted by the liver & the rest appear in urine as urobilinogen.



Types and etiology

Three types are recognized.

1. Haemolytic (prehepatic) jaundice:

- due to excessive RBCs destruction in various types of haemolysis
- characterized by :
 - elevated unconjugated bilirubin .
 - increased stercobilinogen (faecal) and urobilinogen (urinary)
 - Bile salts do not accumulate in the serum.

2. Hepatocellular (hepatic) jaundice

- Mechanism :

- Liver dysfunction → inability to transform unconjugated bilirubin to conjugated → ↑ unconjugated bilirubin level in blood
- Presence of intrahepatic cholestasis → some conjugated bilirubin is reabsorbed → ↑ conjugated bilirubin level in blood.
- Bile salts levels may also rise.

- Causes :

- Viral hepatitis.
- Decompensated liver cirrhosis.
- Bacterial infections as septicaemia and pyaemia.
- Drugs as: Phenothiazine compounds, certain diuretics, testosterone derivatives, oral contraceptives, oral antidiabetic agents, halothane and arsenicals (injure the cellular excretion mechanism → intrahepatic cholestasis)

Common causes of obstructive jaundice

1. CBD stone.
2. Cancer of head of pancreas
3. CBD strictures following iatrogenic injury at cholecystectomy

3. Obstructive (posthepatic) jaundice

- is due to obstruction somewhere in the biliary pathway →
 - a) Conjugated bilirubin reabsorbed to the blood & excreted in urine.
 - b) Bile salts will accumulate in the blood.
 - c) Faecal stercobilinogen and urinary urobilinogen levels are low.
- Causes :
 - a. in the lumen of the bile ducts as:
 - i- calculi
 - ii- parasites as ascaris or fasciola .
 - b. in the wall of the bile ducts :
 - i. Congenital biliary atresia.
 - ii. Inflammatory stricture as with sclerosing cholangitis or 2^{ry} to an impacted stone for a long time.
 - iii. Traumatic stricture (iatrogenic following cholecystectomy, choledocholithotomy or instrumental procedures on the CBD)
 - iv. Malignant stricture.
 - c. outside the bile ducts
 - i- carcinoma of the head of pancreas → obstruct the lower part of the CBD
 - ii- mass of metastatic lymph nodes at the porta hepatis → obstruct the hepatic ducts

Clinical picture

I-History :

- 1) The age and sex of the patient may give a hint to the diagnosis.
- 2) History of drug intake, recent injections and blood transfusion.
- 3) Color of urine and stools.

	Urine	stool
Haemolytic jaundice	normal	Dark
Obstructive & hepatocellular jaundice	Dark	Pale

- 4) Pruritis → absent in haemolytic jaundice.
- 5) Recurrent attacks of haemolytic crisis → haemolytic jaundice.
- 6) Depth of jaundice.
 - a- mild degree → haemolytic jaundice
 - b- severe degree → hepatocellular & obstructive jaundice .
- 7) Course of jaundice
 - a- fluctuant in → Calcular jaundice
 - b- Progressive in → malignant jaundice .
- 8) Pain.
 - a. In haemolytic type → abdominal pain during a haemolytic crisis.
 - b. Calcular obstructive jaundice → upper abdominal pain.
 - c. Malignant obstructive jaundice → painless but may be accompanied by dull aching epigastric pain referred to the back.

II-Examination

A) General examination

- 1- pallor
- 2- pyrexia
- 3- the degree of jaundice
- 4- Virchow's lymph nodes
- 5- cachexia
- 6- stigmata of liver insufficiency
- 7- bleeding tendency
- 8- edema of the lower limbs.

age & sex of the patient importance in diagnosing Jaundice

1. Neonatal period
→ a- physiological jaundice ,
b- Rh incompatibility,
c- neonatal hepatitis,
d- congenital syphilis
congenital biliary atresia.
2. In children →
haemolytic anaemias should be suspected.
3. In middle aged females → calcular jaundice
4. In elderly males → malignant obstructive jaundice

- Signs and symptoms in obstructive jaundice:

1. Jaundice
2. Dark urine
3. Clay colored stools {**acholic stools**}
4. Pruritus

- Ascites may be present in advanced cirrhosis or in metastatic malignant lesions.

- Jaundice is easily recognizable when:

- a. the conjugated bilirubin concentration in the serum reaches 2-3 mg/100 ml or
- b. the unconjugated bilirubin level is 3-4 mg/100 ml

B) Abdominal examination.

- 1- in haemolytic anaemia → hepatomegaly and splenomegaly.
- 2- In calcular obstructive jaundice → negative abdominal examination
- 3- In malignant obstructive jaundice → the liver and gall bladder are commonly palpable.

Investigations

A) Laboratory

1. Blood picture.

- In haemolytic jaundice → i) anaemia.
ii) Special tests to detect various types

2. Serum bilirubin.

- In haemolytic jaundice → ↑ the unconjugated bilirubin level
- In obstructive jaundice → ↑ the conjugated bilirubin
- In hepatocellular jaundice → ↑ both types of bilirubin.

3. SGOT (AST) and SGPT (ALT).

- In haemolytic jaundice → normal level of transaminases
- In obstructive jaundice : i) early stages → normal level
ii) with prolonged obstruction → ↑ enzymes level (dt parenchymatous damage)

- In acute hepatitis → high level
- In cirrhosis → moderate increase

4. Alkaline phosphatase.

- In haemolytic jaundice → normal level
- In hepatocellular jaundice → may be slight increase
- In obstructive jaundice → moderate increase (particularly in malignant obstruction)

5. Prothrombin time and concentration

- In haemolytic jaundice → normal
- In both hepatocellular and obstructive jaundice → prolonged PT & diminished PC

6. Serum albumin

- Low in patients with cirrhosis or after prolonged malignant cachexia.

7. Faecal stercobilinogen

- In haemolytic jaundice → high
- In hepatocellular and obstructive jaundice → low

B) Imaging

1. Abdominal ultrasound. (the 1st investigation)

- in obstructive jaundice → dilatation of intrahepatic biliary radicles

2. CT scan

- In patients suspected of having abdominal malignancy as carcinoma of the head of pancreas → show :

- a- The site and extent of the tumour
- b- Invasion of adjacent structures
- c- Metastatic lymph nodes
- d- liver deposits.

3. MRCP

4. Endoscopic ultrasound.

5. ERCP

6. PTC

To differentiate hepatocellular from obstructive jaundice
→ give a course of IV vitamin K for a few days:

- a. In obstructive
→ the prothrombin parameters will improve
- b. in hepatocellular
→ no improvement

- ERCP → used in obstructive jaundice with lesion involving the **lower** end of the CBD as ampullary carcinoma.

- PTC → used in obstructive jaundice with lesion involving the **upper** end of the CBD as postoperative stricture or carcinoma of the hepatic ducts

Common Bile Duct Stones

Pathology

Formation

1-In most cases, a stone in gall bladder → slopes through the cystic duct → reach the CBD.

2-Primary stones in the CBD are rare and occur when there is prolonged stasis and infection (brown pigment stones)

Effect

1. No effect if the stone remains floating in CBD without obstruction.

2. Passage to the duodenum.

-A small stone < 3mm → pass spontaneously through the sphincter of Oddi → acute pancreatitis.

3. Obstruction of CBD →

a. Obstructive jaundice

b. Dilatation of the bile duct and intrahepatic biliary radicles,

c. Prolonged obstruction → high pressure in the bile ducts → bile secretion by the liver stops → the bile ducts become full of mucous (**white bile**).

d. Complications:

i. Bleeding tendency.

Bile salts fail to reach the bowel → failure of absorption of fat-soluble vitamins including vitamin K → deficient synthesis of coagulation factors

ii. Cholangitis.

- Due to infection by Gram-negative bacilli.

-It leads to severe infection of the *liver* or even septicaemia.

iii. Septicaemia and hepatorenal failure.

-Absence of bile salts in the intestine allows absorption of bacterial endotoxins → may fail to be filtered by Kupffer cells

-Endotoxins cause renal vasoconstriction and glomerular & Peritubular fibrin deposits → renal failure → followed by liver failure

iv. Acute pancreatitis

-occurs if the stone impacts in the lower part at the common channel of pancreatic duct and CBO

v. Biliary cirrhosis (uncommon)

- In prolonged cases where obstruction is intermittent or incomplete.

Clinical picture

Type of patient: middle aged females

Symptoms

1. Asymptomatic (Silent stones) If there is no obstruction

2. Pain.

- Site: in the right hypochondrial and epigastric regions

- Character: recurrent attacks of severe dull aching pain

-refer: to the right scapular region or even to the back.

- Associated symptoms: nausea and vomiting.

- Bile duct stones are called choledocholithiasis

-Incidence

15% of patients with calculi cholecystitis have stones in the CBD

- Septicaemia and hepatorenal failure are the usual causes of death in unrelieved CBO obstruction

- There is no relation between the size of the stone and the development of jaundice.

- A tiny stone impacted in the ampulla may cause jaundice, while a large calculus in a dilated CBO may not cause obstruction.

- **Charcot triad** (in cholangitis) show recurrent attacks of: a. Pain
b. Jaundice
c. Fever and rigors.

-Reynold's pentad

a. Charcot triad
b. Altered mental status
c. Shock

3. Jaundice.

- Course: slowly progressive & fluctuating
- Degree: not reach a severe degree.
- Cause: stone impaction in the narrow distal part of the CBD.
- Fate: stone either: a) slips to the duodenum
b) floats back up to the dilated CBD → relief of jaundice.

4. Urine → dark & frothy 5. Stools → pale 6. Pruritis.

Signs

1. itching marks
2. signs of bleeding tendency (purpura & ecchymosis)
3. The gall bladder is usually not palpable (chronic inflammation → fibrosis → thick wall → resisting pressure of retained bile)

Also rise in pressure is low due to intermittent calcular obstruction

Investigations

A) Laboratory

I- Blood Picture

-During an attack of cholangitis → polymorph nuclear leucocytosis

II- Liver function tests

1. bilirubin → raised (mainly the direct)
- Usually bilirubin does not exceed 10 mg/dL & may fluctuate.
2. Alkaline phosphatase (the most sensitive)
- If other causes of raised alkaline phosphatase are excluded,
3. Prothrombin time and concentration
- disturbed due to failure of absorption of vitamin K.
4. Serum aminotransferases → ALT (SGOT) & AST (SGPT)
- may show slight rise especially if there is cholangitis.
5. Gamma glutamyl transferase and 5-nucleotidase
- are both maximally elevated in obstructive jaundice

B) Imaging

1-Abdominal ultrasound :

- Dilatation of intrahepatic biliary radicles
- The diameter of the CSD is increased (normal 4 to 7mm)
- Chronically inflamed gall bladder with calculi (points that obstruction is due to calculi but not conclusive evidence)
- Presence of stones in the CBD in a good number of cases.
- A mass in the head of the pancreas may be detected.

2- Abdominal CT

- demonstrates lesions of the pancreas or metastatic lymph nodes.

3- ERCP

- detect lesions of the ampulla and a biopsy can be taken.
- Visualized both the CBD and pancreatic ducts .
- A stone will appear as a filling defect
- should not be done during an attack of cholangitis because the rise of pressure during injection of the contrast material → severe sepsis (unless a therapeutic procedure to drain bile is planned)

4- MRCP

Good diagnostic but not therapeutic

Courvoisier's law

A palpable distended gall bladder in a patient with obstructive jaundice , the cause of obstruction is not stone → mostly malignant obstruction due to carcinoma of the head of pancreas
Explanation :
malignancy
→ complete continuous obstruction → a much higher rise in pressure → distends a healthy thin-walled gall bladder → easily felt large palpable gall bladder

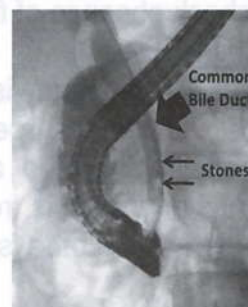
Differential diagnosis

a) Hepatocellular jaundice due to:

1. Viral hepatitis.
2. Drug toxicity.

b) Malignant obstructive jaundice:

1. Carcinoma of head of pancreas
2. Cholangiocarcinoma of bile ducts



Aim

1. To relieve biliary obstruction by removal of stones from CBD. (1st)
2. To remove the gall bladder, the source of CBD calculi.

Preoperative preparation

Admission to hospital for a few days for preparation :

1. correct the coagulation abnormalities
by injections of vitamin K1
2. prevent the possibility of renal failure by :
 - a) Adequate hydration by IV fluids
 - b) IV mannitol if urine output is not satisfactory
 - c) Oral bile salts (diminish the liability to endotoxaemia)
3. Guard against liver cell failure
broad spectrum antibiotics if there is evidence of cholangitis.

Definitive treatment

the favored treatment is endoscopic extraction of CBD calculi followed by cholecystectomy (by laparoscopy or open).

A-If ERCP is available

• Procedure

1. Diathermy (electrocautery) → do endoscopic sphincterotomy.
2. The stone(s) is removed by **Dormia** basket or a balloon catheter.
3. A large stone can be fragmented before removal either by :
i- mechanical ii- electrohydraulic iii- laser lithotripsy.

complications.

1. Bleeding (in 2-9% of cases)
- This may be due to coagulation defects.
- PT should be checked before the procedure & give vitamin K IV.
2. Acute cholangitis (in 1-3% of cases)
- It may progress to septicaemia and death especially in cases with failed bile duct clearance after sphincterotomy.
3. Pancreatitis (in 1-4% of cases)

B-If ERCP fails to clear CBD of stones, treatment is by operative exploration of CBO (choledocholithotomy) and removal of the stones together with cholecystectomy.

• Procedure:

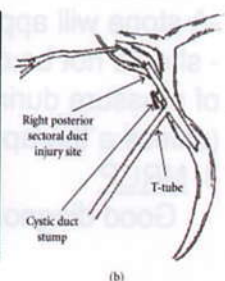
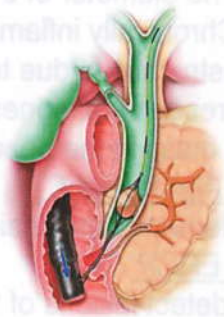
1. The CBD is opened and the stones are extracted.
2. A T-tube is inserted in the CBD which is closed around it.
The long limb of the tube is brought outside the patient → help in the drainage of bile in the early post-operative period.
3. A T-tube cholangiogram can be performed after closure of CBD to check absence of filling defects (completion T-tube cholangiography)
4. Cholecystectomy is then performed.
5. **10 days** after surgery ,a cholangiogram through the T-tube is performed and if there are no residual stones and there is free flow of the contrast to the duodenum → tube is removed

In the absence of cholangitis, calcular obstructive jaundice is not an emergency

- If the patient is debilitated with Suppurative cholangitis → temporary relief of biliary obstruction can be achieved by PTD or stent insertion at ERCP and definitive management postponed until the patient is fit for surgery

- If endoscopic drainage fails, rapid surgery is mandatory to achieve biliary drainage

- If endoscopic drainage fails, rapid surgery is mandatory to achieve biliary drainage



Strictures of Bile Ducts

I-Congenital biliary atresia → see pediatric surgery chapter

II- Traumatic Strictures

Etiology

- follows cholecystectomy (usually) or choledocholithotomy (less)
It may be due to:

1. Complete ligation of the common hepatic or CBD.
2. Narrowing of the duct due to its inclusion in a ligature.
3. Devitalization of the duct due to rough dissection.
4. Ischaemia of the duct

Clinical picture

-present by either: a) biliary fistula
b) jaundice following cholecystectomy

Investigations

1-ERCP 2- MRCP 3- PTC

Localize site of the stricture → usually at the upper part of CBD

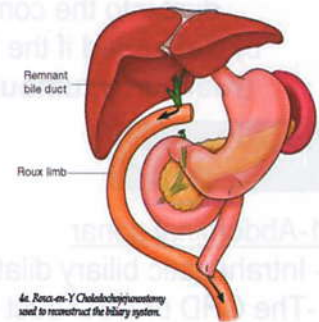
Treatment

- 1- A trial of dilatation and stenting may be tried
 - 2- if this fails →operative correction performed.
- The remaining stump of the common hepatic duct (if present) or the left hepatic duct is anastomosed to a Roux-en-Y loop of jejunum.

Causes of strictures in bile duct:

- 1-Congenital
- 2-Traumatic
- 3- Inflammatory→
Sclerosing cholangitis
- 4-neoplastic

- The commonest cause of CBD stricture is iatrogenic injury at cholecystectomy



III- Sclerosing Cholangitis

Etiology

Unknown

Pathology

- The intra-and extrahepatic ducts are involved by multiple strictures separated by normal or dilated segments

Clinical picture

-At first presents by obstructive jaundice
-if untreated → lead to liver failure

Investigations

MRCP or ERCP →show multiple strictures with a beaded appearance

Treatment

- Anastomosis of a dilated segment of the bile duct if present to a loop of jejunum.
- If there is no available dilated segment suitable for anastomosis → candidates for liver transplantation

IV-Neoplastic stricture (carcinoma of bile ducts)

Predisposing factors

- 1- ulcerative colitis
- 2- primary sclerosing cholangitis
- 3- parasitic infestation by clonorchiasis
- 4- calcular disease
- 5- choledochal cyst

Clinical picture

Type of patient : common in elderly males

Symptoms

- 1- Obstructive jaundice (in 90% of patients).
- 2- Pain, cholangitis and anorexia (less common)

signs

- 1- Hepatomegaly.
- 2- Gall bladder will be :
 - a) **Distended** If the tumour is below the insertion of the cystic duct into the common bile duct
 - b) **Collapsed** if the tumor is at the porta hepatis (**Klatskin's tumour**) → (an exception to Courvoisier's law).

Investigations

1-Abdominal sonar

- Intrahepatic biliary dilatation.
- The CHD or CBD duct will be dilated down to the site of the lesion.

2- ERCP is useful in low strictures.

3- PTC

- Will show intrahepatic biliary radicles.
- Show the proximal dilated portion of the CHD in high lesions

4- MRCP

Treatment

A) Low strictures:

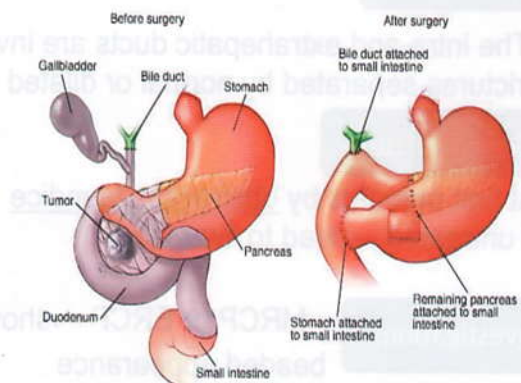
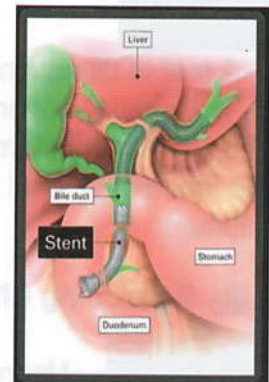
- If operable → pancreaticoduodenectomy (**Whipple operation**).
- If inoperable →stenting

B) High strictures:

- If operable → excision and Roux-en-Y hepaticojejunostomy
- If inoperable → stenting by ERCP or PTD
“percutaneous transhepatic drain”

In case of obstructive jaundice, if GB is palpable , it is most probably non calcular (malignant) with exceptions :

- 1- Associated chronic cholecystitis
- 2- Malignant obstruction above cystic duct (Klatskin's tumor)
- 3- Previous cholecystectomy



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Carcinoma of the Gallbladder

Pathology

- gallstones are present (In > 90% of cases)

- Microscopic picture :

- squamous cell carcinoma
- adenocarcinoma
- a mixture of the two.

-Spread

direct → of the liver and porta hepatis

by lymphatics → to the hilar lymph nodes

blood (venous) → to the liver



Clinical picture

type of patient : more common in elderly female

symptoms

1- usually diagnosed after pathological examination of the gall bladder that was removed because of chronic calculous cholecystitis.

2. acute cholecystitis

If the tumour obstructs the cystic duct.

3. Obstructive jaundice

If the bile duct is obstructed by : a) the tumor

c) metastatic lymph nodes in the porta hepatis.

Signs

- Mass in the right hypochondrium

Investigations

- 1- Ultrasound
- 2- CT scan
- 3- MRI

} will visualize the gallbladder, liver and lymphatic metastases

Treatment

A-If the diagnosis is suspected during surgery,

-it should be confirmed by frozen section and if positive radical cholecystectomy is performed.

-This operation includes:

- 1- Excision of the gall bladder.
- 2- Excision of a wedge of the underlying liver.
- 3- Clearance of lymph nodes overlying the bile duct.

B-if the lesion is fairly advanced and inoperable (the usual)

- Internal stenting to relieve jaundice.
- The prognosis for this lesion is very poor

Functions of the liver

1. Formation and secretion of bile.

- Bile salts, cholesterol & phospholipids formed in hepatocytes.
- Hepatocytes also pick up unconjugated bilirubin from the blood stream and conjugate it to glucuronic acid to form conjugate Bilirubin.
- Bile reaches the intestine through the common bile duct that opens in the duodenum.
- In the intestine it emulsifies fat prior to its digestion, allows absorption of fats, vitamin K and other fat soluble vitamins, regulates intestinal motility, and provides the natural brown color of stools.
- Bile salts have mild antiseptic properties and prevent the intestinal bacteria from being absorbed into the portal circulation.
- Lack of bile salts in the intestine in obstructive jaundice → vitamin K deficiency → hypoprothrombinaemia and hemorrhagic tendency.
- Its lack, also is accompanied by septicaemia, shock, and Renal failure (which are the common causes of death)
- Most of the bile salts in the intestine are absorbed in the terminal ileum, reaching the liver to be re-excreted in bile → enterohepatic circulation.

2. The site of carbohydrate, protein, and fat metabolism.

- Glycogen is formed and stored in the liver then converted to glucose and released to the circulation in case of need.
- Proteins as albumin, prothrombin, and fibrinogen are formed.
- is also where glucose is formed from proteins by the process of gluconeogenesis
- Urea is formed by deamination of amino acids.
- A great part of energy is generated in the liver from the Krebs cycle, part of which is stored as adenosine triphosphate (ATP).

3. Metabolism of many drugs and hormones, e.g. oestrogens.

4. Removal of ammonia from the portal blood.

5. The storage house of glycogen, vitamin B₁₂, vitamin A, iron and copper.

6. Reticuloendothelial cells clear the blood from the bacteria that can escape from the intestine to the portal circulation

- The liver receives 1.5 L of blood per minute, of which 2/3 supplied by the portal vein while 1/3 supplied by the hepatic artery
- Because of its higher oxygen content, the hepatic artery supplies 50% of the oxygen requirements.

- Regenerative hyperplasia

- The liver cells have a remarkable capacity for regeneration by hyperplasia when part of it is damaged by disease or is surgically resected
- This ability is greatly diminished in cirrhotic livers.
- Surgical resection of a part of a Cirrhotic liver carries a bad prognosis because of the diminished capacity of Regeneration & because of the poor function of the remaining part

Studies of the Liver

A. Laboratory tests

1. Liver function tests

Test	Normal range
Tests which assess the synthetic functions of the liver - Serum albumin - Prothrombin time (PT) - Partial thromboplastin time (PTT)	35-50 gm/L 10-15 seconds 35-40 seconds
Tests which assess the excretory functions of the liver - Alkaline phosphatase - 5'-Nucleotidase - Gamma-glutamyl transferase	30-110 IU/L 10-50 IU/L
Tests which denote liver cell injury - Serum aspartate aminotransferase (AST) - Serum alanine aminotransferase (ALT)	5-40 IU/L 5-40 IU/L
Serum bilirubin total and direct is a test of the synthetic and excretory functions • Total bilirubin • Direct bilirubin	0.3-1.0 mg/100 ml 0.35 mg/100 ml

2. Tests for viral markers (hepatitis A, B, C, D and E)

3. Tumour markers: Alpha fetoprotein and CEA

B. Imaging of the liver

1. Ultrasonography

- The first choice imaging investigation of the liver because it is simple, non-invasive, reliable, not expensive & easily repeated
- It is particularly useful for detecting focal lesions, and allows for guided biopsy

2. Triphasic computed tomography (CT scan) and dynamic magnetic resonance imaging (MRI)

- Visualize the parenchyma and adjacent tissues clearly

3. Arteriography

- Is now losing popularity in favour of the previously mentioned non invasive techniques
- Useful in the diagnosis and management of liver tumours.

4. Isotope scanning of the liver

- was previously in common use to detect space occupying lesions of the liver.
- It has been nearly replaced by ultrasonography or CT scan

Alkaline phosphatase may be raised in bony or intestinal problems.

C. Liver biopsy

Techniques

1. The percutaneous technique (The standard method)
 - To reduce bleeding, the prothrombin time should not be prolonged > 3 seconds above the control value
 - The platelet count should be > 80,000/uL
 - Even so, it is safer to cross match two units of blood to be ready in case of bleeding.
 - The procedure is done under local anaesthesia through the right midaxillary line just below the upper limit of liver dullness
 - The commonly used needles are the aspiration (Menghini) & the cutting (Tru-cut) types.
 - To biopsy a focal lesion, passage of the needle is guided by ultrasound or CT scan.
2. Laparoscopic biopsy.
3. Liver biopsy at laparotomy.
 - In diffuse liver disease → obtain a deep wedge biopsy from the lower border of the right and the left lobes.
 - An intraoperative needle biopsy from the depth of liver substance further increases the diagnostic accuracy.

Liver Trauma

- Liver injuries are commonly associated with affection of other intra or extra abdominal organs → commonly: The ribs, pleura, lungs, colon and spleen
- The prognosis after treatment depends on associated injuries

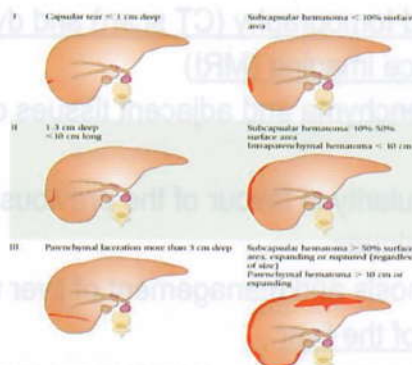
Etiology

1. Accidental trauma that is either:
 - a) blunt → as in road traffic accidents (RTA)
 - b) Penetrating as with stabs and bullets
2. Iatrogenic → is increasing with the rising popularity of invasive investigations as percutaneous liver biopsy & PTC
3. Spontaneous rupture of the liver is an extremely rare that may happen with eclampsia or hepatic tumours.

Pathology

Type of injury

1. Small subcapsular haematoma.
2. Small superficial tear or tears.
3. Large subcapsular or intrahepatic haematoma.
4. Large deep tear or tears
5. Shattered liver parenchyma which may include a whole lobe
6. Vascular injury → **the most difficult to control is that of the main hepatic veins because of the difficult access.**



- Liver biopsy indications:

1. Target biopsy of focal lesions that are suspected to be 1st or 2nd tumours is sometimes indicated.
2. Liver transplant assessment.
3. A preoperative histological diagnosis is essential & the technique can also help in the early detection of postoperative transplant rejection.

Contraindication →
Bleeding diathesis

-Haemangiomas and hydatid cysts should not be biopsied

-The liver is the 2nd common solid abdominal organ to be injured.
- It is preceded only by the spleen both account for **75%** of all blunt abdominal injuries
- In bullet injury the damage is directly proportionate to its velocity, so the most harmful are the high velocity missiles of rifles

Consequences

1. Bleeding (the main danger)
 - should be the main concern of the surgeon.
 - Most liver injuries stop bleeding by the time of exploration but some of them cause death from blood loss.
2. Haematuria
 - Liver haematoma sometimes communicates with a torn bile duct → blood to trickle down the biliary passages to GIT

Clinical picture

1. History of trauma.
2. Abdominal pain.
3. Abdominal tenderness and rigidity (due to parietal peritoneal irritation, either by blood or escaping GIT secretions)
4. Massive bleeding presents with the picture of hemorrhagic shock

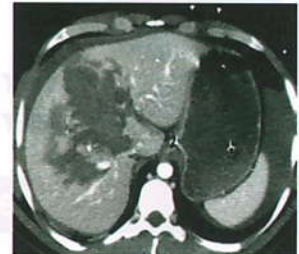
Investigations

1. chest x-ray
 - The presence of lower rib fractures →suspects liver affection
2. Diagnostic peritoneal lavage (DPL), ultrasound or CT scan
 - Are done in suspected cases → discover minor bleeding
 - These tests are particularly useful in the unconscious patient as it is difficult to assess the abdomen.
3. Laparotomy
 - Systematic exploration for penetrating abdominal trauma.

Treatment

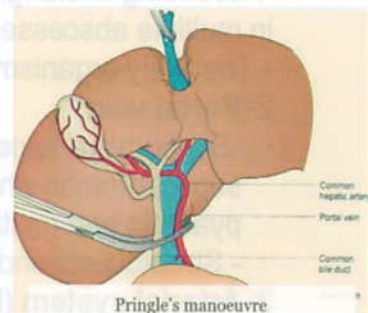
- 1- Management of polytraumatized patient (ATLS)
- 2- Haemodynamically stable patients with no evidence of peritonitis → treated conservatively by repeated examination, CT scan may be repeatedly performed
- 3- Haemodynamically unstable patients , have peritonitis or deteriorate under conservative treatment → need laparotomy

- Liver injuries draw attention to their presence because of intraperitoneal haemorrhage.
- Chest stabs as high as the 4th intercostal space can transfix the diaphragm and injure the liver (And/or the spleen)



Principles of surgical management:

1. Adequate exposure by a longitudinal incision that can be extended to the chest in case of need.
2. Thorough systematic exploration of the abdomen to assess the liver affection and to detect other intra-abdominal injuries.
3. Priority is for arrest of bleeding.
 - most small liver tears stop bleeding by the time the abdomen is explored so no treatment required
 - liver haemorrhage can be controlled by a combination of temporarily packing the bleeding area and the application of Pringle's manoeuvre (which is application of a vascular clamp to the free border of the lesser omentum or holding it between two fingers, to occlude the hepatic artery and the portal vein for a period up to 20 minutes)
4. The lessened rate of bleeding allows the surgeon to visualize and ligate the injured vessels



5. Dealing with different types of injury

A) Suturing liver tears (whenever possible) should be avoided because it is likely to leave a space for accumulation of haematoma that may infect or communicate with intrahepatic bile ducts.

-**however:** it is resorted to if control of bleeding vessels is not possible in deep tears → tying sutures over pedicled omentum (Deep transverse mattress sutures using special liver needle)

-A haematoma is explored to ligate the damaged vessels and ducts and to excise the dead tissues then left open for drainage.

-A lobe that is shattered beyond salvage → excision of this lobe.

B) Firm packing of inaccessible and difficult bleeding areas, e.g., the hepatic veins

-May be the only method for temporary arrest of bleeding.

-The patient is transferred to a specialized centre where the pack is removed in the operating theatre & deal with the injury

C) Multiple intraperitoneal drains

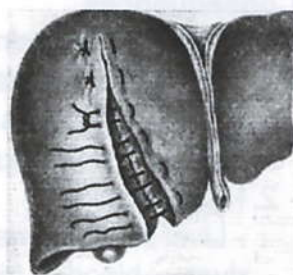
-Are placed to guard against collections of blood and bile

D) Prophylactic antibiotics are prescribed

Prognosis

-The mortality of liver injury **15-20%**.

-It gets worse if other major organs are injured



- Suspect liver injury in patients with fractures in right lower ribs
- Minor liver injuries can be treated conservatively –
- Pringle's maneuver is useful for temporary control of hepatic bleeding during the operation
- Perihepatic packing is very useful as a last resort to stop bleeding in serious hepatic injuries

Infections of the Liver

Pyogenic liver abscess

This is a serious highly fatal disease. The key to improving the results of its treatment is an early diagnosis.

Etiology

A) Sources of infection

1-Biliary tract (cholangitic abscess).

-This is the commonest of all sources.

-Ascending cholangitis caused by bile duct obstruction results in multiple abscesses of the liver.

- The likely organism is *E. coli* or other gram negative bacilli.

2-Portal vein.

-Suppurative appendicitis or colon diverticul may induce septic thrombo-phlebitis of the portal vein radicles → portal pyaemia and multiple liver abscesses.

- Streptococci and anaerobes are the common organisms.

3-Arterial system (haematogenous abscess).

The source may be bacterial endocarditis, tonsillitis, intravenous drug misuse or osteomyelitis.

-Staphylococcus aureus is the usual causative organism.

4- Idiopathic.

-The source of infection is not possible to trace.

Infections of the liver:

1. Viral hepatitis
2. Pyogenic liver abscess.
3. Amoebic hepatitis and abscess.
4. Hydatid disease of the liver.
5. Hepatic Schistosomiasis.

- Today, fewer patients present with portal pyaemia probably because of early diagnosis of intraperitoneal sepsis and the widespread use of potent antibiotics

B) Predisposing factors

a. The immunocompromized patient, e.g., diabetes mellitus, leukaemia, chronic illness, the elderly, the alcoholics, and transplanted patients receiving immunosuppressive therapy.

b. An already existing liver lesion

As hydatid cyst, amoebic abscess, or haematoma (rarely)

Complications

- **Direct extension** to the pleura, lung, pericardium, or peritoneum

Clinical picture

Symptoms:

- Fever and its constitutional manifestations, toxemia with right hypochondrial or lower chest pain

Signs

- Abdominal examination → tender hepatomegaly (however, may be absent and the patient just presents with PUO)

- Features of the causative lesion are evident, e.g. the Charcot's triad of cholangitis, or the pain of acute appendicitis

Investigations

A) laboratory

a. Leucocytosis.

b. Anemia and high ESR.

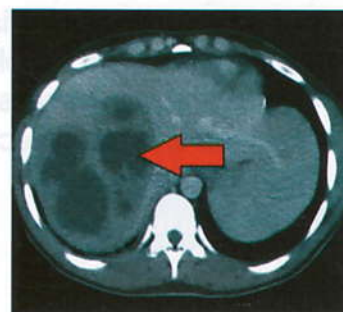
c. Low serum albumin.

d. High alkaline phosphatase and transaminases level.

e. Elevated serum bilirubin in cholangitis or multiple abscesses.

B) Imaging.

Ultrasound or CT scan → diagnose and localize the abscess.



Treatment

1- Broad spectrum antibiotics are prescribed

2- As for any abscess the treatment is drainage of pus.

- Now depend on percutaneous guided drainage of pus.

- The procedure is simple that is done under local anaesthesia, where ultrasound or CT is used to direct the needle towards the abscess cavity

- The abscess is aspirated then a tube drain (**Pig tail**) is inserted

- This procedure has replaced the standard method of open or laparoscopic surgical drainage.

-Incidence of amoebic hepatitis:

1. Commoner than the pyogenic abscesses in developing countries particularly those lying in the tropical & subtropical regions.
2. The low standards of hygiene coupled with high humidity favour amoebiasis infestation.

Amoebic Hepatitis and Abscess:

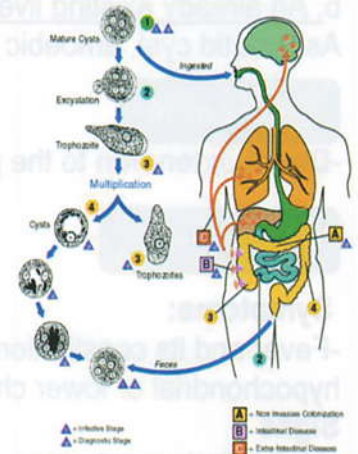
Etiology

- Due to infection with the protozoal parasite *Entamoeba histolytica*.

- The condition is a complication of amoebic colitis.

Gross picture

- 1- The trophozoites invade portal blood from the colon & migrate up to reside in the liver
 - 2- The right lobe (especially the **posterosuperior segment** which is the commonest site) of the liver is *affected* more than the left.
- This is explained by the observation that portal blood from the right side of the colon drains mainly to the right lobe of the liver while that from the left colon drains to the left lobe
- As amoebic colitis *affects* mainly the right colon, it is expected that amoebic hepatitis affects mainly the right side.
- 3- The parasite starts a process of liquefactive necrosis and when there is heavy infection → the necrosis results in the formation of an abscess that is usually single and unilocular.
 - 4- Pus is usually sterile with a **brown chocolate** color and sometime pinkish → likened to '**anchovy sauce**'.
 - 5- The abscess has a shaggy wall that harbours the amoeba.



Complications

- 1- Secondary bacterial infection.
 - 2- Rupture into the pleura, lung, pericardium, or peritoneum.
- When the abscess ruptures upwards → lung abscess rather than empyema because 2 layers of the pleura are obliterated

Clinical picture

Symptoms

- 1- Upper abdominal pain.
- 2- Low-grade fever

(In contrast to the high-grade fever seen with a pyogenic abscess)

- 3- Anorexia, nausea and weight loss.
- 4- A clear history of an attack of dysentery not always obtained.

Signs

- 1- The patient is pale and looks toxic → an earthy look.
- 2- Tender hepatomegaly, there may be tenderness in the lower right intercostal spaces
- 3- Chest-examination → RT basal lung abscess or RT pleural effusion

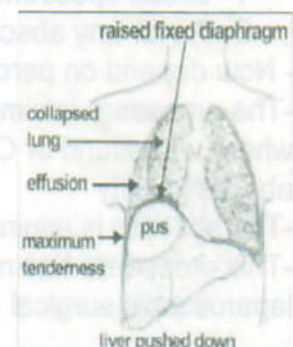
Investigations

A) Laboratory

- 1- Positive stool analysis and serological tests
 - Are useful in diagnosis in nonendemic areas.
 - In countries where amoebic colitis is prevalent, these tests are likely to be positive in a high percentage of the population.
- 2- Blood picture.
 - There is commonly leucocytosis and anaemia

Differential diagnosis:

1. Pyogenic liver abscess.
 2. Hepatocellular carcinoma
- in black Africans assumes a rapidly progressive course with pain and constitutional symptoms that simulate those of an amoebic abscess



3- Therapeutic test

- The high success of metronidazole treatment and improvement in the general and local conditions after 3 days of treatment confirms the diagnosis

B) Imaging

- 1) ultrasound or CT scanning (mainly)
 - Can detect the number, site and size of the abscess.
- 2) A chest X-ray
 - Detect pleural effusion and pulmonary collapse.
 - Also reveals an elevated right copula of the diaphragm

Treatment

1- Conservative treatment

- Is highly successful.
- Metronidazole is the drug of choice
(800 mg three times daily for 7 -10 days)

2- Ultrasound-guided percutaneous aspiration

- Is needed for abscesses that fail to respond to treatment within 72 hours and for large abscesses.
- Aspiration is done by a large bore - spinal needle under local anaesthesia.
- The site of aspiration depends on the site of the abscess
 - a) If anterior → insert needle below the costal margin anteriorly
 - b) If posterior → inserted in the 10th intercostal space posteriorly
 - Few days later ultrasound is repeated and if the abscess cavity has recollected and is > 5 cm → aspiration is repeated

3- Open drainage

Indications: a) Presence of secondary infection.

b) If the abscess is pointing.

c) If aspiration is difficult because of multilocular abscess or the presence of thick pus.

- Metronidazole is diagnostic and therapeutic

- Although diagnosis is by isolation of the parasite, patients with clinical signs of amoebic abscess will be treated empirically with metronidazole and investigated only if they do not respond

- As most of amoebic abscesses are in the postero-superior segment of the RT lobe, open drainage which is rarely needed, is done through the bed of the 12th rib posteriorly through an extrapleural approach.

Hydatid Disease of the Liver

Geographical distribution

- Prevalent in sheep-rearing parts of the world
- In the Middle East the disease is commonly seen in Iraq, Yemen, and Libya but is sometime also met within Egypt

Etiology

Causative agent

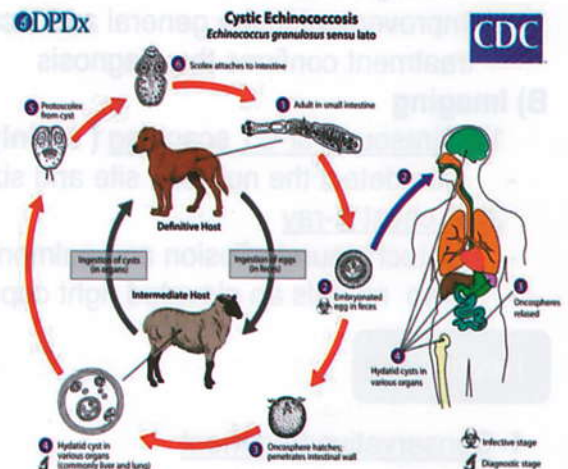
- By the tapeworm *Echinococcus granulosus*.

Echinococcus



Life cycle

- 1- The adult worm lives in the intestine of the dog
(**The definitive host**)
- 2- Ova are passed in the dog's stools and ingested by sheep as they feed on contaminated grass.
- 3- In the sheep's stomach → ova hatch, penetrate the wall and enter the portal venous system to lodge and form cysts mostly in the liver, and less frequently in the lungs or brain.
- 4- Sheep are considered to be the 2^{ry} host
- 5- The parasite's life cycle is continued when dogs eat liver of dead infected sheep
- 6- Man may be accidental 2^{ry} host through vegetables or hands that become contaminated with dog's excreta.



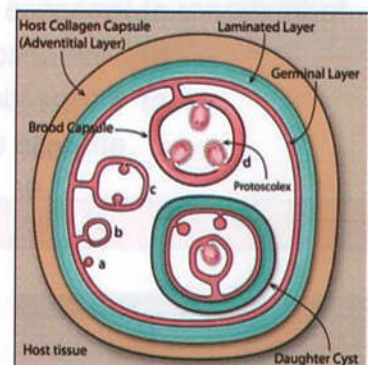
Pathology

Gross picture

- 1- The liver may be the site of a single or multiple hydatid cysts.
- 2- The cyst fluid is usually colorless and clear but may be yellowish if the cyst communicates with the bile ducts.
- 3- Scolices are found in this fluid → may cause a severe anaphylactic reaction if it gets to the circulation

Microscopic picture (double-layer cyst wall)

- 1- Germinal layer (endocyst)
 - The inner thin layer
 - Is the living part of the parasite that secretes the hydatid fluid
 - Shows folds forming broad capsules that contain the heads of future worms (scolices)
- Daughter cysts may form inside the main cyst.
- 2- laminated membrane (ectocyst)
 - the outer layer
 - formed by parasite from germinal layer
 - separated from adventitia by plane of cleavage (This plain lost in liver infections)
- 3- Adventitia (pericyst)
 - Formed by the host as reaction to intrusion of parasite



Fate and complications

The fate is quite variable.

- a) The majority of cysts continue to enlarge gradually.
- b) In others the parasite dies and the cyst is calcified.

Complications that endanger the patient's life (small proportion)

- a) Secondary infection
- b) Rupture of the cyst in the biliary passages causing jaundice
- c) Rupture in the peritoneal cavity → dissemination and an anaphylactic reaction.

Clinical picture

- 1- May be asymptomatic for years and discovered accidentally with Ultrasonography that is done for another purpose
- 2- Usual presentation is chronic right upper quadrant pain & hepatomegaly
- 3- Rarely the disease presents by one of its complications
- 4- In 70% of cases, the cyst is felt as a well-defined painless fluctuant swelling

Investigations

A) Laboratory

- 1- Blood picture: may show eosinophilia
- 2- Intradermal Casoni's test
 - The disadvantage:
 - a) Once positive, it remains so for the rest of the patient's life.
 - B) Gives false positive results in 40% of cases.
 - Is now replaced by the more sensitive haemagglutination, (ELISA) test or complement fixation test → detects hydatid antibodies in the serum.

B) Imaging

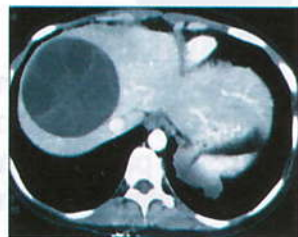
- 1- Ultrasound or CT scan
 - Shows the well-circumscribed cyst (or cysts).
 - In endemic area, liver cyst is usually caused by hydatid disease.
 - Daughter cysts may be also visualized.

Treatment

- 1- surgical removal (except for small asymptomatic cysts)
 - a. Preoperative estimate of the number and sites of the cysts
 - b. The danger with excision is the possibility of hydatid fluid spillage into the peritoneal cavity→
 - i) Risk of peritoneal implantation of the scolices forming other cysts
 - ii) Absorption into the circulation → a life threatening anaphylactic reaction.
 - c. When the liver cyst (or cysts) is approached, it is surrounded by dark green towels that are moistened with a scolicial agent such as hypertonic sodium chloride or povidone iodine (betadine).
 - The dark green color is suitable background to visualization any spilled scolices
 - d. The cyst is first aspirated and hypertonic sodium chloride is injected to make it three quarters full.
 - e. The overlying liver substance and the surrounding adventitia are incised and the double layered cyst is easily enucleated.
 - f. Small cysts need no further treatment
 - g. The cavity left by a large cyst is filled with pedicled omentum.
2. PAIR (Puncture, Aspiration, Injection of hypertonic saline and Reaspiration)
 - Recent line of treatment
 - A substitute or complementary to surgery.

The 2001 World Health Organization (WHO) classification of hepatic hydatid cysts is used to assess the stage of hepatic hydatid cyst on Ultrasound, is useful for deciding on its appropriate management, depending on the stage of the cyst.

- There are 6 classes of hydatid cyst in U/S (CL, CE1-5).
- CE 5 entails the presence of calcification in the wall of cyst = the cyst is inactive and infertile thus no treatment required



- Risks→ spillage, anaphylactic shock, haemorrhage or sclerosing cholangitis if the cyst is communicated to the biliary tree

3. Medical treatment:

- With mebendazole or albendazole
- Not a good substitute for surgery but is used in:
 - a. Treatment of the elderly frail patients.
 - b. Treatment of recurrent cases.
 - c. Some advise pre and postoperative use to prevent recurrence.
- The usual dose of mebendazole is 400-600 mg 3-times daily for 2 weeks preoperatively and 3-months postoperatively

LIVER CIRRHOSIS

Definition

- Condition in which the whole liver is replaced by multiple nodules separated from one another by anastomosing sheets of fibrous tissue
- Cirrhosis is the end result of the following pathological processes
 1. Long continued loss and necrosis of liver cells.
 2. Persistent inflammatory reaction.
 3. Regeneration of the liver lobules in an irregular manner with loss of architecture → large number of nodules.
- place of portal tracts and hepatic veins are irregular in the nodules
- 4. Extensive fibrosis.
- 5. Fibrosis and architectural distortion → interfere with blood flow → continuous loss of liver cells even in the absence of the original insult which caused the cirrhosis.

Etiology

1- Post-viral.

- Now the most common cause of cirrhosis in Egypt and it can occur in association with bilharzial periportal fibrosis.
- 10% of patients who get virus B and 50% of patients who get virus C hepatitis → not recover completely and develop chronic active hepatitis.
- There is piece-meal necrosis of the liver (destruction of the liver cells at the interface between connective tissue and parenchymal cells)
- Lymphocytes or plasma cells infiltrate between liver cells → cirrhosis of the macronodular type.
- This type occurs at a younger age than alcoholic cirrhosis
- Its clinical progress is rapid.
- There is also a higher incidence of liver cell carcinoma.

2- Alcoholism.

- Common cause especially in western countries (high & continuous consumption)
- Hepatotoxic level is a daily dose of 100 gm of alcohol

LIVER CYSTS:

Include the following:

1. Simple cyst.

- a solitary non-parasitic cyst which is believed to be of congenital

2. Polycystic liver disease (PCLD).

- a congenital disease usually associated with AD polycystic kidney disease.

- Rarely associated with hepatic fibrosis of liver failure.

3. Abscesses.

4. Neoplastic cysts.

These are either due to:

- a) Central necrosis of liver tumours
- b) True intrahepatic neoplastic cysts (rare) (cystadenoma or cystadenocarcinoma).

- CEA, mucin and cytology from the cyst fluid are required to differentiate benign from malignant cysts.

- Treatment is liver resection.

- Cirrhosis entails necrosis, regeneration & fibrosis.

- It was previously thought to be an irreversible disease, now appears that with treatment of the underlying insult, (e.g. chronic hepatitis C, alcohol abstinence, haemochromatosis) can be some reversal of fibrosis.

-Alcohol alone produce cirrhosis but associated nutritional deficiency aggravate its toxicity.

- Chronic alcoholism → lead to fatty liver → 1/3 of them will progress to alcoholic hepatitis → 1/3 of them complicate with cirrhosis.

-More in males usually between 40-70 years of age.

- Usually in the early stages is micronodular type but later it may show the mixed or the macronodular varieties.

-Progresses more slowly than most other types

-Abstinence of alcohol intake may lead to improvement.

3- Biliary cirrhosis.

- 2^{ry} to long-continued cholestasis due to the harmful effect of retained bile on the hepatocytes.

- There are two types:

a. Primary biliary cirrhosis is of unknown etiology

-There is lymphocytic and plasma cell infiltration of the small intra-hepatic bile ducts → fibrosis of the portal tracts and cholestasis → irregular loss of liver cells and nodular regeneration (complete the picture of cirrhosis.)

- supposed to be due to an immune reaction → can be diagnosed by the detection of anti-mitochondrial antibodies and by liver biopsy.

b. Secondary biliary cirrhosis

-Due to prolonged mechanical obstruction of the larger biliary passages as in congenital biliary atresia, sclerosing cholangitis or post-operative biliary stricture

-Biliary obstruction → accumulation of bile in the hepatocytes, bile canaliculi and kupffer cells.

-A progressive inflammatory reaction develops in the portal tracts followed by fibrous septa linking up adjacent portal tracts.

4- Congenital cirrhosis.

- 2^{ry} to a group of metabolic abnormalities and include:

a. Wilson's disease (Hepatolenticular degeneration).

-is AR disease in which increasing amounts of copper accumulate in and damage the liver, the kidneys, the lenticular nuclei and eyes.

-The end result will be liver cirrhosis, Kayser Fleischer rings in the cornea and muscular tremors and spasticity.

b. Haemochromatosis.

- Excess deposits of iron in hepatocytes, Kupffer cells and portal tract macrophages.

- Increased amounts of lipofuscin are also present in hepatocytes.

-Excess iron → stimulate fibrous tissue formation → cirrhosis.

-Excess iron deposits occur also in the pancreas → diabetes.

c. Alpha-antitrypsin deficiency and galactosemia

5. Nutritional

-Protein deficiency can lead to cirrhosis.

-The liver in these patients is susceptible to some toxic agents which do not affect the normal liver.

6. **Cryptogenic cirrhosis** → no definite cause.

7. **Cardiac cirrhosis** is due to:

a. Long-standing right-sided heart failure.

b. Budd-Chiari syndrome.

c. Veno-occlusive disease

Bilharzial Periportal Fibrosis

Pathology

Gross appearance

- In the early stages the liver may be of a normal size
- enlarged if there is:
 - a) Fatty infiltration of liver cells
 - b) Hyperplastic regenerating nodules.
- As the disease progresses → the liver shrinks due to loss of liver cells and extensive fibrosis.
- The surface is nodular
- the cut surface reveals that the whole liver is replaced by rounded nodules separated by fibrous tissue
- Cirrhosis is classified according to the gross appearance into
 - a) **Micronodular type**
 - The nodules are of the same size and about 3 mm in diameter.
 - b) **Macronodular type**
 - The nodules are of variable size and up to 1 cm in diameter.
 - c) **Mixed type.**

Microscopic appearance

- The nodules show loss of the normal architecture
- The portal tracts and hepatic veins lost their regular spacing.
- There are foci of liver cell atrophy and foci of hyperplasia and hypertrophy so that the cells are enlarged and include binucleate forms.
- There is extensive fibrous tissue septa between the nodules composed of fine or dense collagen fibres.
- The portal tracts & hepatic veins are included in fibrous tissue.
- Lymphocytes and plasma cells infiltrate the connective tissue and less commonly the parenchymal nodules.

Gross and microscopic appearances of bilharzial periportal fibrosis is classified into 2 types depending on the type of portal tracts involved

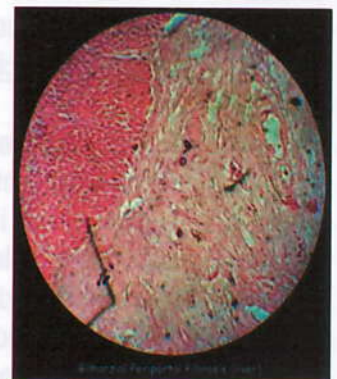
(a) Fine bilharzial periportal fibrosis.

- Few ova are deposited in the small portal tracts.
- The external surface of the liver is nodular, each measure up to 15 mm in diameter and are separated from each other by depressions in which the capsule is opaque and thickened by fibrous tissue.
- Microscopic examination reveals strands of fibrous tissue enclosing partially or completely one or more of the liver lobules.
- Few bilharzial ova may be within the fibrous tissue.

(b) Coarse bilharzial periportal fibrosis.

- Large number of ova is deposited in the large portal tracts.
- Gross fibrous thickening of the affected tracts occurs.
- The liver is often reduced in size.
- The surface shows multiple shallow depressions separating slightly raised areas.

-This is not true cirrhosis.
-A large number of ova laid in the terminal radicles of the mesenteric vessels fail to engage in the walls of the vessels and form emboli in the portal blood.
- The ova reach the intrahepatic radicles of the portal vein and escape in the portal tracts → produce bilharzial granuloma → fibrosis → excess fibrosis of the portal tracts (simulating cirrhosis)
-As the criteria of cirrhosis are not present, bilharzial fibrosis is not considered as true cirrhosis



- The cut surface shows the characteristic thickened large portal tracts (**pipe-stem cirrhosis**).
- Microscopy may show bilharzial worms in the portal veins
- There is no gross damage to the liver lobules in bilharzial fibrosis.
- Only some pressure atrophy of the liver lobules may occur in the neighborhood of the thick portal tracts.
- sequel of bilharzial periportal fibrosis is obstruction of the portal vein radicals in the portal tracts (**Presinusoidal obstruction**)

Clinical sequelae

- the patient will suffer from two major problems
- (a) Hepatocellular insufficiency due to diminution of functional capacity of the liver.
- Cirrhosis may remain silent for some time and is discovered only after routine liver function tests or abdominal ultrasound.
- In some patients the functional reserve of the liver is adequate to make them look fine, but an insult to the liver as in :

- | | | |
|------------------------------|---|------------------------------|
| 1- GIT haemorrhage | } | severe parenchymatous damage |
| 2- the use of narcotic drugs | | |
| 3- bacterial infection | | |
| 4- anaesthesia | | |

- (b) Portal hypertension due to interference with the portal blood flow

Cirrhotic patients will usually elicit one or more of the following

1. Anorexia with loss of weight and ill health.
2. Spider naevi.
 - These are located in the face, neck and upper arms, each consists of a central arteriole from which radiate small vessels.
3. Palmer erythema.
 - The hands are warm, the thenar and hypothenar eminences are bright red
4. Testicular atrophy and gynaecomastia
 - occur in males due to failure of the liver to inactivate oestrogens.
 - In females there may be menstrual irregularities, breast atrophy or secondary amenorrhoea (the mechanism is unknown)
5. Bleeding tendency
 - leads to epistaxis, bleeding gums and easy to form haematoma
 - There is disturbance in the synthesis of all coagulation factors except factor VIII in addition to thrombocytopenia due to portal hypertension or hypersplenism.
6. Hyperdynamic circulation with warm hands and increased cardiac output is probably due to;

a) Vasoactive substances	b) Hypervolaemia
--------------------------	------------------
7. Jaundice
 - Mild or moderate jaundice is present in late cases of cirrhosis.
 - It is a bad prognostic sign.
8. Ascities→ multifactorial
 - a. **Hypoalbuminaemia** as the liver is the only site of albumin synthesis (when albumin level drops < 3 gm/100 ml → ascities)

Portal hypertension alone cannot cause ascities

b. **Salt and water retention** due to high levels of aldosterone, oestrogens and antidiuretic hormone.

c. **Increased lymphatic exudation** from the surface of the liver due to lymphatic obstruction

9. Hepatic encephalopathy.

- represents a combination of neurologic symptoms and signs accompanying advanced decompensated liver disease and/or extensive portal-systemic shunting.
- The exact etiology not known
- The patient develops apathy, lack of awareness, lethargy and disorientation (may be coarse flapping tremors & muscular rigidity)
- The condition may progress to deep somnolence and finally coma.

10. Common associations.

- a. Chronic duodenal ulcer.
- b. Chronic pancreatitis
- c. Enlargement of the parotid glands.
- d. Dupuytren's contracture.
- e. Primary liver carcinoma (HCC develop in 10% of cirrhotics)

Treatment

Unfortunately established cirrhosis is an irreversible pathology.

- complete abstinence from alcohol.
- avoid intake of any drugs or anaesthetics that are hepatotoxic.
- High carbohydrate intake.
- Vitamins.
- **The only curative treatment is liver transplantation**

Portal Hypertension

Definition

Portal hypertension is present when the portal vein Pressure exceeds 20 mmHg (25-30 cm of water).

Etiology

Mainly due to ↑resistance to porta venous flow

I- Pre-hepatic causes

1. Congenital malformation of the portal vein
 - it is replaced by a cavernomatous mass.
2. Neonatal umbilical sepsis leads to thrombophlebitis of the umbilical vein → obliteration of the portal vein
3. Portal vein thrombosis secondary to :
 - a) intra -abdominal sepsis
 - b) use of oral contraceptives
 - c) with liver cirrhosis (rare)
 - d) hepatocellular or pancreatic carcinoma
 - e) Chronic pancreatitis.

II- Intrahepatic causes

1. sinusoidal and post sinusoidal
 - Liver cirrhosis.
2. presinusoidal
 - Bilharzial periportal fibrosis.

-Suggested causes of hepatic encephalopathy:

- a) High levels of ammonia
- b) False neurotransmitter as octapamine, mercaptans
- c) Other amines in the blood and cerebrospinal fluid.
- d) Defective ratio between branched chains to aromatic amino acids

- The normal portal pressure equals 7 mmHg (8-12 cm of water).
- The average blood Flow to the liver is about 1500 ml/minute of which 2/3 from the portal vein and 1/3 from the hepatic artery
- In most of patients with pre-hepatic portal hypertension the liver will be normal

- Portal hypertension in cirrhotic patients dt:
 - (a) Diminution of the total vascular bed by obliteration, distortion and compression of sinusoids.
 - (b) Compression of the tiny radicles of portal and hepatic veins by excessive fibrosis.
 - (c) Development of multiple arteriovenous shunts between the branches of the hepatic artery and of the portal vein

III- Post-hepatic obstruction

1. Budd-Chiari syndrome (occlusion of hepatic vein by)
 - a. Thrombosis which may be spontaneous or 2^{ry} to polycythaemia or the use of oral contraceptives
 - b. Malignant invasion
2. Veno-occlusive disease (obstruction of the hepatic venules)

Sequelae and clinical picture

1- Porto-systemic collaterals.

There are multiple sites of porto-systemic anastomoses but they are collapsed under normal conditions due to the lower pressure of the portal circulation

-With progressive portal hypertension → collaterals become engorged to divert the blood away from the portal circulation.

-The important sites of these collaterals are:

Site	between	
	Portal circulation	Systemic circulation
a. The lower part of the oesophagus and fundus of the stomach (oesophageal & gastric varices)	tributaries of the left gastric and short gastric veins	oesophageal veins which drain into the azygos and hemiazygos veins
b. In the anterior abdominal wall (caput Medusae)	the paraumbilical vein	the superior and inferior epigastric veins
c. The lower rectum and anal canal (anorectal varices)	Superior haemorrhoidal vein	middle and inferior haemorrhoidal veins
d. Retroperitoneum	the superior and inferior mesenteric veins	posterior abdominal wall and subdiaphragmatic veins

2- Splenomegaly due to:

- a. Reticuloendothelial hyperplasia
 - In bilharziasis due to absorption of bilharzial toxins
- b. congestion of spleen
 - 2^{ry} to portal hypertension
 - May reach the right iliac fossa.
 - It predispose to :
- i. recurrent attacks of **splenic infarction** → severe stitching pain → heal by fibrous tissue → adhesions between the spleen and the diaphragm and the abdominal wall

ii. secondary hypersplenism

3-Congestion of the whole GIT

- Leads to anorexia, dyspepsia, indigestion and malabsorption leading to abdominal discomfort which is wrongly attributed to Splenomegaly

-Venous hum can be auscultated on caput Medusae due to the increased blood flow.

-If haemorrhoids occur in a patient with portal hypertension they are usually primary.

-Portal HTN itself can lead to hypersplenism
 -In bilharziasis, splenomegaly is due to 1st RES hyperplasia then congestion later
 - Ascites may cause an umbilical hernia.
 -Infection of ascitic fluid may lead to 1^{ry} (spontaneous) bacterial peritonitis

4-Ascites

-causes (multifactorial):

a) Portal hypertension

-alone cannot cause ascites, It only localizes the filtration of fluid into the peritoneal cavity.

b) Hypoalbuminemia

- When albumin level drops < 3 gm/100 ml, ascities usually develops

c) Salt and water retention

-due to high levels of aldosterone, oestrogens & antidiuretic hormone

d) Increased lymphatic exudation

-from the surface of the liver due to lymphatic obstruction

Investigations

The aims are:

1-Assessment of the functional capacity of the liver

- Done by liver function tests which will reveal:

a. Prothrombin time & concentration (**Most sensitive**) are disturbed

b. Hypoalbuminaemia (liver is only site of albumin synthesis)

c. ALT and AST are moderately raised

2- Detection of oesophageal varices

 by either:

a. Fibreoptic upper endoscopy

b. Barium swallow → visualize varices in 90% of cases

-They appear as multiple, smooth, rounded filling defects

c. Duplex scan can show dilated portal vein and collaterals.

3-Detection of splenic sequestration and hypersplenism

a. Blood picture reveal anaemia, leucopenia thrombocytopenia or pancytopenia.

b. Bone marrow examination reveal hypercellularity.

c. Radioactive isotope studies

- using the patient's own RBCs tagged with Cr^{51} → diminished half life of RBCs and increased radioactivity over the spleen.

-The spleen /liver radioactivity index is increased.

4-Diagnosis of the etiology of liver disease:

a. Immunological tests for hepatitis markers.

b. Liver biopsy after assessment of prothrombin time and concentration

Esophageal and gastric varices:

-Are present in the subepithelial, submucous & periesophageal layers

-Their enlargement →

Erosion of overlying mucosa → bleeding (in form of haematemesis, melaena or bleeding per rectum "if massive")

-Bleeding ppt. by high portal pressure, trauma of food or reflux of gastric juice → oesophagitis

- Bleeding oesophageal varices is **the commonest cause of**

haematemesis in Egypt (especially in bilharziasis or cirrhosis)

-patient with oesophageal varices may bleed from another lesion, e.g. gastric erosions or peptic ulcer.



Child -Turcotte-Pugh (CTP) classification

Is used to assess the parenchymatous functions of the liver

	Points		
	1	2	3
Encephalopathy	None	Grade 1-2 (or precipitant induced)	Grade 3-4 (or chronic)
Ascites	None	Mild/ moderate (diuretic responsive)	Severe (diuretic-refractory)
Bilirubin (mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
PT (sec prolonged)	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3

-CTP obtained by adding the score for each parameter:

A = 5-6 points

B = 7-9 points

C = 10-15 points

-Patients with CTP:

- class A can have major surgery
- class C cannot have major surgery

Treatment

I- Silent varices (primary prevention is important)

- Lifetime risk of a first-time variceal bleed in a cirrhotic patient is **30%** with mortality rate up to **50%**.
- Endoscopic screening for varices should be done in all cirrhotics.
- Primary prevention with a non-selective beta-blocker (propranolol or nadolol) decreases the risk of a first variceal bleed by up to **40%**.
- if large varices present (especially if they have stigmata of bleeding "red streaks on the variceal surface"), banding is indicated.

II- Management of patients with actively bleeding varices

1. Admission.

- Even minor haematemesis (may be followed by massive bleeding)

2. Resuscitation.

- A wide bore cannula is inserted.
- A blood sample is taken
- Restoration of the patient's blood volume should be rapid.

3. Correct coagulopathy.

- Vitamin K is given IV
- Fresh frozen plasma may be needed.

4. Prevent encephalopathy.

-Blood in the intestine will be fermented to ammonia and other nitrogenous product → absorbed but they by-pass the liver through the portosystemic collaterals → a high level of nitrogenous products in circulation → hepatic encephalopathy

(Even those which reach the liver are not completely detoxified due to disturbed liver function)

-should evacuate blood from GIT to prevent its fermentation by:

- Repeated enemas or colonic lavage.
- Oral lactulose
- Neomycin **0.5 gm every 4 hours** (reduce the bacterial flora)

- Patients with portal hypertension will belong to one of three categories:

1. Portal hypertension with silent varices.
2. Actively bleeding oesophageal varices.
3. Patients with history of bleeding oesophageal varices.

- A blood sample is taken for:

- a) Complete blood picture
- b) Liver function tests
- c) Coagulation studies
- d) cross-matching for at least 4 units of blood

- Overexpansion of the circulation is avoided as it may lead to heart failure, ascites or renewed bleeding.

-Morphine & Pethidine are contraindicated in these patients

5. Stop bleeding.

-Measures to stop the bleeding are arranged in order of priority. If one method fails, the next is used

A) Urgent upper GI endoscopy

- To confirm the source of bleeding (**diagnostic**)
- For Endoscopic sclerotherapy or band ligation (**therapeutic**)
- Once the patient is resuscitated→ taken to endoscopy department & an oesophagogastric endoscopy is performed under diazepam.

-If the varices are:

a) Not bleeding at examination → look for other sources as PU

b) Bleeding → they are injected.

-There are two methods for injecting varices:

a) Intravariceal method: 5 mls of ethanolamine oleate injected into each varix just above the oesophagogastric junction → thrombosis

-For gastric varices intravariceal injection of histoacryl → solidifies after contact with blood.

b) Perivariceal method: small quantities of aethoxysclerol (0.5 ml) injected multiply alongside each varix → perivascular fibrosis.

- Both methods may be combined.

-Injection must be repeated at 2 weekly sessions until the varices are obliterated.

-Band ligation (**now the first choice**)

1. Encircle each varix by a tight band → thrombosis of the veins.

2. It is as effective as sclerotherapy

3. Number of sessions is less than sclerotherapy.

B. Drugs

I. Vasopressin

-Produces vasoconstriction of the arterioles of the splanchnic circulation → reduces the portal pressure.

- Dose: 0.2 unit/kg wt in 200 ml of 5% dextrose over 20 minutes.

-Disadvantages are:

i- Colicky abdominal pains

ii- diarrhea

iii- angina pains (so contraindicated in elderly)

- produce temporary control of bleeding, to prolong its action it is combined with glycine (Glypressin).

II. Somatostatin can lower the intravariceal pressure.

C-Balloon tamponade

- used as a temporary measure if bleeding is too profuse to permit endoscopic treatment then arrange for repeat endoscopy or emergency TIPSS or surgery.

- Types :

a) Sengstaken-Blackmore tube

b) Linton tube

-The gastric balloon is inflated first by **200 ml of air** and pulled upwards to press the gastric fundus.

-If bleeding continues, the oesophageal balloon is inflated with pressure not exceeding **40 mm Hg**.

- Effective in controlling bleeding in **80-90%** of cases.

-Oral lactulose is a disaccharide sugar which is fermented by the intestinal flora to lactic acid→ combines with ammonia

-It also has a mild laxative effect that helps clear blood from the bowel.

- Injection of bleeding varices is more difficult than in elective cases as the oesophagus may be full of blood but the result is very gratifying as bleeding stops in **90%** of cases

- Side effects of injection

sclerotherapy include

i. Retrosternal discomfort (few days)

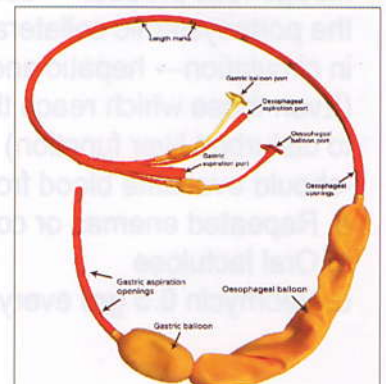
ii. Fever.

iii. Repeated injections can cause an

oesophageal ulcer → bleeding.

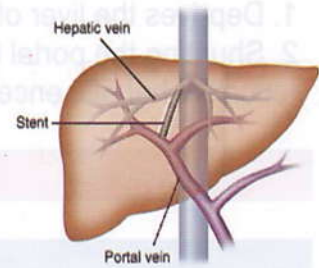
iv. Repeated sclerotherapy → fibrosis & stricture.

v. penetration of the oesophagus → mediastinitis (rare)



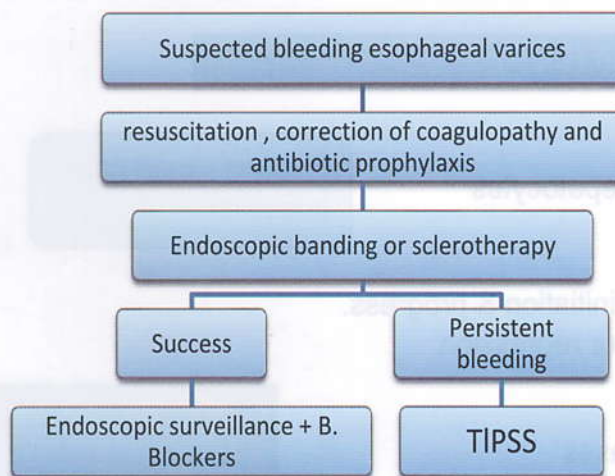
D-Transjugular intrahepatic porto-systemic shunt (TIPSS)

- Recommended procedures if all the previous measures fail
- Under fluoroscopic guidance a guide wire is passed through the internal jugular vein → hepatic vein → through liver → until portal vein branch.
- A stent is passed along the guide wire until it lodges inside the liver creating a shunt between a portal and hepatic vein
- Very effective in the control of variceal haemorrhage.
- Complications:
 - 1) Perforation of the liver capsule → fatal haemorrhage.
 - 2) Porto-systemic encephalopathy in 40% of patients.
 - 3) Shunt occlusion 50% after one year.
- The results of shunt operations and esophageal stapling in patients with bleeding varices are not satisfactory and have a high mortality.



Disadvantages of balloon tamponade include:

- I. Discomfort to the patient.
- II. The patient cannot swallow his saliva → aspiration of saliva (There are tubes which contain a special channel for aspiration)
- III. Liability to cause oesophageal ulceration or stricture
- iv. Once the tube is deflated, there is liability to rebleeding in 60 -80% of patients.



III-Treatment of patients with history of bleeding oesophageal varices

- Bleeding from esophageal varices represents the major threat to cirrhotic patients, usually massive
- Once the patient started bleeding, further attacks can occur
- Management:

1. First choice is repeated sclerotherapy or band ligation until the varices are obliterated
2. If sclerotherapy or esophageal banding failed → elective surgery is indicated provided that they are fit.

Operations for portal hypertension

(A) Shunt operations:

- The idea is to lower the portal pressure by shunting the portal blood away from the liver.
- So long as the portal pressure remains in the normal range, the varices will collapse and bleeding stops.

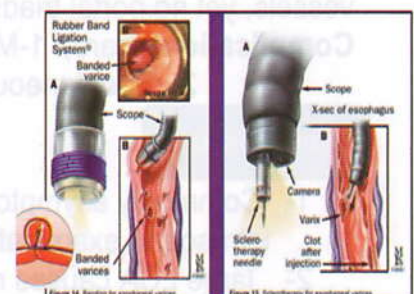
-Examples:

a) Porta caval operation:

The portal vein is anastomosed to the inferior vena cava

b) Splenorenal operation:

Where the splenic vein is anastomosed to the left renal vein



Bleeding from varices is related to:

1. Size of varices
2. Bad liver functions (child C)
3. Wedged hepatic venous (WHV) pressure >10 mmHg

-Disadvantages:

1. Deprives the liver of portal blood flow→ accelerates liver failure.
2. Shunting the portal blood completely away from the liver leads to recurrent hepatic encephalopathy in **30-50%** of patients.

Liver Tumours

Benign	Malignant
1-True neoplasms :Liver cell adenoma (LCA) 2- Non-neoplastic lesions a) Haemangioma b) Focal nodular hyperplasia	I-1 ^{ry} : a)Primary hepatocytes: Hepatocellular carcinoma (HCC) b)Bile ducts: Cholangiocarcinoma c)Mixed: Cholangiohepatoma II-2 ^{ry} : metastasis from GIT , pancreas or breast cancer

Liver cell adenoma (LCA)

Definition

This lesion is a true benign neoplasm of hepatocytes

It occurs almost only in women

Etiology

- Contraceptive pills are incriminated in its initiation & progress.
- Cessation of pill intake can result in tumour regression

Pathology

Gross picture: 1- It is multiple in 1/3 of cases

2-Is soft, well-circumscribed & light yellow in color

Microscopic picture

Is formed of sheets of regular hepatocytes with thin-walled vessels, yet no portal triads

Complications (rare): 1-Malignant transformation

2- Spontaneous rupture → internal haemorrhage

Clinical picture

- 1- Commonly asymptomatic (discovered incidentally at ultrasound examination or at laparotomy)
- 2- large LCA causes right upper quadrant pain

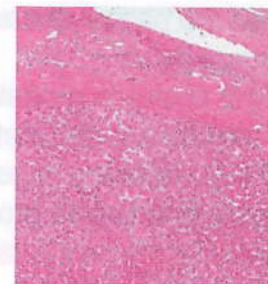
Investigations

Imaging: by ultrasound, CT scan and /or MRI

Laboratory: Liver function tests and alpha fetoprotein within normal

Treatment

- 1- the contraceptive pill intake is discontinued and regularly check change of size of tumor
- 2- Liver resection is indicated if:
 - a) doubt exists as to the possibility of malignancy
 - b) Large (>5 cm) symptomatic tumours.



Haemangioma

Pathology

- They are of the cavernous type and may attain a large size.
- They are usually harmless
- Occasionally rupture → serious bleeding

Diagnosis

- 1- Ultrasound → focal lesion
- 2- CT scan with contrast

Treatment

The natural history is towards involution and calcification

- 1- Asymptomatic haemangiomas (even diagnosed at Laparotomy) should not be resected
- 2- Excision only for lesions that produce pain or bleeding



-Haemangiomas are the commonest benign tumours of the liver
-Suspicion of a haemangioma is a contraindication for biopsy



Focal nodular hyperplasia (FNH)

Definition

This is a harmless lesion of the liver whose main importance is the difficult differentiation from other focal lesions

Pathology

Gross picture

- A nodular firm mass, slightly paler than the normal liver
- Has prominent surface vasculature
- The cut section shows a stellate scar with radiating fibrous septa that divide the lesion into lobules

Microscopic picture

- It resembles cirrhosis with regenerating nodules
- The central scar and fibrous septa are apparent

Diagnosis

- 1- Triphasic CT scan : reveal the pathognomonic **stellate scar** This differentiates it from hepatocellular carcinoma or metastases
- 2- Dynamic MRI scan

Treatment

- 1- Most cases require no treatment
- 2- If malignancy suspected → explore and excise

-Etiology of FNH is unknown but is believed to be caused by congenital abnormalities of the liver blood vessels
→ some liver cells to receive more blood → grow into a tumour like



Primary Liver Cancer

Etiology

I- HCC

1. Infection with hepatitis B virus (HBV), or hepatitis C virus (HCV).
2. Liver cirrhosis of any cause (the risk depend on type)
- The highest incidence is post-hepatitic followed by alcoholic
3. Aflatoxin ingestion
- Formed by the fungus *Aspergillus flavus* which grows on grains that are stored in moist warm conditions as in Africa

II- Cholangiocarcinoma

- Infestation with the liver fluke ***Clonorchis sinensis*** that cause Asiatic cholangitis

Pathology

I-HCC

Gross appearance

- The tumour is yellow in color and arises in a cirrhotic liver.
- Has different appearances:
 - a) **Massive form** → forming a localized mass.
 - b) **Nodular form.** → multiple nodules
 - c) **Diffuse form** → diffuse infiltration throughout the liver

Microscopically

- Formed of malignant hepatocytes with little stroma but with high vascularity derived from the hepatic artery.
- The cells possess acidophilic cytoplasm and large nuclei with acidophilic nucleoli.
- In well differentiated → cells are similar to normal liver cells
- In anaplastic → less similar

Complications

1. Spread by the lymphatic and venous routes → porta hepatis nodal enlargement, peritoneal nodules, and less commonly lung deposits.
2. Spontaneous rupture → subcapsular haematoma or massive intraperitoneal haemorrhage

II- Cholangiocarcinoma:

- An adenocarcinoma epithelium lining the intrahepatic bile ducts
- By the time tumour is discovered is usually has widely spread

III- Hepatocholangiocarcinoma

- This is a mixed type that behaves like HCC.

IV- Hepatoblastoma.

- Is a form of HCC that occurs in children
- Is named so because of the similarity to fetal liver cells.

Clinical picture

- Depend on the *race*, development of the country and tumour size.
- 1- Small lesions may be accidentally discovered through an abdominal ultrasound examination that is done for another purpose.

Epidemiology:

-Type of patient:
commoner in men

-HCC

a) Is the main primary liver malignancy
b) Prevalent in the Far East and in equatorial African nations.

c) The world's highest incidence in Mozambique.

- Cholangio-carcinoma

a) Arising from the intrahepatic bile ducts
b) Less common than HCC
c) Highest incidence in the Far East.

-90% of HCC are related to viral hepatitis (B or C) or to liver cirrhosis.
≈ 10% of patients with liver cirrhosis will develop HCC.

- Benign tumors are more common in females

-1st liver cancer is commoner in males



2. Deterioration of health of a known cirrhotic (**common**)
3. HCC in black Africans grows rapidly & degenerates → pain, jaundice, fever and tender mass (commonly mistaken for an amoebic liver abscesses)
5. Late cases → pain, jaundice, ascites, hepatomegaly, anorexia, loss of weight and massive intraperitoneal haemorrhage.

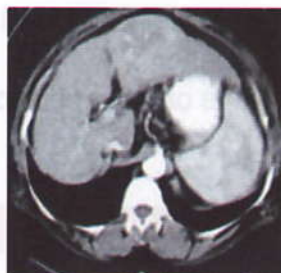
Investigations

I- Laboratory tests

- 1- Serum alkaline phosphatase → elevated
 - The rest of liver function tests may show cirrhosis if present
2. Serum alpha-fetoprotein (AFP)
 - A tumour marker that is present in the serum of the fetus
 - The normal adult level is 0 to 10 ng/mL
 - The serum level of this marker is highly elevated with HCC and with testicular teratoma.
 - A level above 200 ng/mL is suggestive (> 400 ng/mL is present in large or rapidly growing tumors)
 - Benign liver diseases elevate level of serum AFP but lesser
 - Serum AFP returns normal level after a successful resection.
 - Re-elevation in the follow up period signifies a recurrence

II- Imaging

- 1- Ultrasound, CT scan or MRI
 - Detect the site and extent of tumour spread in the liver.
- 2- Triphasic (arterial, portal and hepatic) multi-slice CT scan via an IV contrast (**Mandatory**)
 - show the anatomical relations of the tumour to the liver vasculature → early arterial enhancement and early washout.
- 3- Chest x-ray and CT scan to search for chest metastases.
- 4- Intraoperative ultrasound: detect small lesions which may be missed by other imaging modalities.
 - It allows safer resection by identifying major vessels and bile ducts.
- 5- Laparoscopy: to visualize the extent of the tumour (peritoneal nodules can be seen) and perform laparoscopic ultrasound.
- 6- PETscan to detect any systemic metastases



Treatment

- **Surgery is the only hope of cure in patients with HCC.**
- **Either hepatectomy or liver transplantation in suitable patient**
- The presence of cirrhosis poses some problems for patients undergoing resection as:
 - a) Bleeding tendency.
 - b) Poor function of the remaining liver.
 - c) Diminished capacity of liver cell regeneration.

- 90% of HCC occurs in patients that are carriers of hepatitis virus or who have cirrhosis

- Prolonged infection with HBV or HCV results in integration of the viral DNA into the host genome resulting into malignancy
- Rapid deterioration of health of a patient with cirrhosis → suspect malignancy
- In areas where HCC is a common tumour an early detection program is recommended where known cirrhotics have an annual ultrasound examination and alpha-fetoprotein estimation.
- An elevation of the tumour marker level or the appearance of a focal lesion warrants further investigations to detect the tumour at its earliest phase.

-The fibrolamellar HCC:

1. Is a variety of the massive type that affects non-cirrhotic young females
2. Has better prognosis
3. Contains numerous fibrous septa which can occur in a normal liver.

Pre-requisites for hepatectomy

- 1- Child class A patients.
- 2- No evidence of extrahepatic spread.
- 3- No vascular invasion or portal vein thrombosis.
- 4- The tumour is confined to one lobe of liver
-in this case the operation is either right or left hemihepatectomy

Pre-requisites for liver transplantation

- 1- Child class C patients.
- 2- No evidence of extrahepatic spread.
- 3- Single lesion < 5 cm or 3 small nodules each < 3 cm
(**Milan criteria** to one lobe of the liver)

Preoperative guided biopsy is not desirable due to:
a) Possible risk of haemorrhage from the highly vascular tumour
b) Seeding of tumour cells

Inoperable cases,

The following palliative options are available:

1-Transarterial chemoembolization (TACE)

- Has benefits of: a) Inducing tumour ischemia
b) Selective chemotherapy.
- The chemotherapy agent is loaded on gelfoam and is injected by angiographic techniques into the hepatic artery →artery blocked →tumour ischemia and drug is selectively delivered to its target.

2- Regional ablation by:

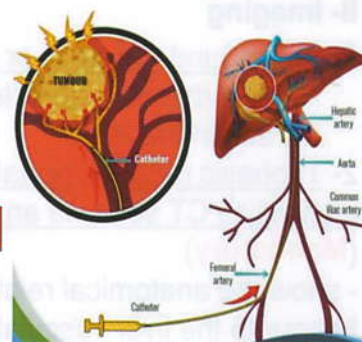
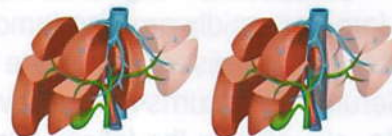
- a) ultrasound guided intra –lesional alcohol
- b) Intra-lesional radiofrequency
- c) Intra –lesional microwave or laser ablation

3- Systemic chemotherapy

a Right hemihepatectomy b Extended right hemihepatectomy



c Left lateral liver resection d Left hemihepatectomy



Liver Metastases

Metastases can reach the liver through either:

1. Portal circulation (**the commonest**)
-Carcinomas of the colon, rectum, pancreas or stomach metastasize primarily to the liver.
2. Hepatic artery.
- In Carcinomas of the lung, breast, kidney, uterus or ovaries
3. Lymphatics.
4. Direct spread from tumours of the gall bladder, stomach or colon.

Pathology

- 1- Liver metastatic nodules are usually multiple, white & umbilicated because of central necrosis
- 2-They are usually adenocarcinomas
- 3- Over 90% of patients have tumour deposits in other organs.

Clinical picture

In addition to the manifestations of 1^{ry} tumour, the patient suffers:

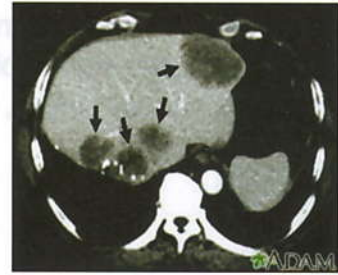
1. Weight loss, fatigue & anorexia.
2. Right upper abdominal pain.
3. Jaundice.
4. Ascites.
5. Palpable hepatomegaly

Metastases may occur at the same time of discovery of The primary tumour (**synchronous**) or months & years after resection of the primary (**meta-synchronous**)



Investigations

- 1- Search for the primary tumour.
- 2- Laboratory tests reveal:
 - a. Anaemia.
 - b. Serum ALP and bilirubin are usually elevated.
3. Imaging by ultrasound, CT scan or MRI → diagnostic and shows the number and sites of metastases



Treatment

- the presence of liver metastases = advanced inoperable tumour.
- The treatment is usually by chemotherapy (palliative)

I- Chemotherapy

- In presence of other organ involvement → Systemic chemotherapy
- If liver is the only organ with metastases → Selective intra-arterial chemotherapy through a catheter that is surgically inserted in the hepatic artery (reduces the systemic side effects of the anti-cancer agents and allows delivery of higher doses to the liver)
- Intralesional alcohol injection or radiofrequency ablation.

II- Liver resection:

The prerequisites:

1. A completely resectable colorectal cancer with no residual tumour.
2. No extrahepatic metastases are found after a thorough search
3. Liver metastases those are resectable with a safety margin:
 - a) A solitary metastatic nodule → removed in a wedge resection.
 - b) Multiple metastases confined to one surgical lobe → removed by a right or left hemihepatectomy.

Differential diagnosis

- 1- Hepatocellular carcinoma.
- 2- Metastasis
- 3- Benign lesions as: haemangioma, adenoma, and focal nodular hyperplasia.
4. Cyst → parasitic or non-parasitic

-The most common
-Primary malignant liver tumour is HCC.
-Benign liver tumour is haemangioma.
- A few selected cases of metastatic colorectal cancer, if localized to one segment, can be treated by liver resection aiming at cure

- Liver resection is done in the same session of the colorectal operation, or later if the liver deposits appear in the course of the patient's follow-up → about 25% of these highly selected patients are cured

Rupture of the spleen

- One of the most frequently injured organ in the abdomen.
- Its injury causes severe blood loss.
- spleen is normally small, hidden by ribs and protected by the thick abdominal muscles.

Etiology

Predisposing factors

- 1- Splenic enlargement → makes it more liable to trauma.
- 2- Diseases of the spleen like malaria which make it friable

Types of trauma

- 1- Blunt abdominal trauma or trauma to lower thoracic cage as in road traffic accidents and falling from a height.
- 2- Penetrating trauma of gunshots or stabbing.
- 3- Operative trauma occurs during an operation on adjacent viscera as during gastric or colonic surgery
- 4- Spontaneous rupture of the spleen is rare.

Pathology

Types of splenic injury

- 1- Subcapsular haematoma.
- 2- Small superficial tears (single or multiple)
- 3- Deep tear (single or multiple)
- 4- Avulsion of a pole of the spleen.
- 5- Complete pulping of the spleen.
- 6- Injury of the vascular pedicle (vessel avulsion or thrombosis)

Complications

- 1- Hemorrhage (intraperitoneal or retroperitoneal or in the pleura if the injury involves the diaphragm)
- 2- Splenic cyst may follow a perisplenic haematoma
- 3- Associated other abdominal or thoracic injuries.

Clinical picture

There are 3 clinical presentations

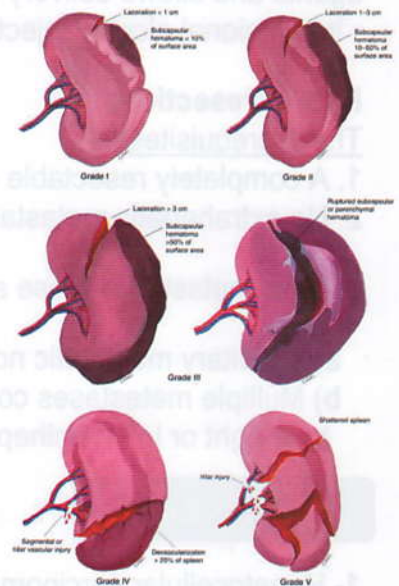
I. **Classical rupture (is the commonest presentation)**

The general manifestations (of internal hemorrhage)

- 1- weak and rapid pulse
- 2- low blood pressure
- 3- dyspnoea
- 4- Increasing pallor

Abdominal examination

- 1- tenderness and rigidity in the left hypochondrium
- 2- Shifting dullness might be elicited.



Special signs

1- Kehr's sign.

- Referred pain in the left shoulder due to irritation of the diaphragm, especially if the patient is in the Trendelenburg position.

2- Cullen's sign.

- Brownish or bluish discoloration around the umbilicus (Occur in about 20% of people who have thin linea alba around the umbilicus through which blood can shine)

3- Ballance's sign (late and pathognomonic)

- Shifting dullness on the right side due to free fluid blood in the peritoneal cavity in the right flank
- Fixed dullness on the left side is due to the presence of intraperitoneal clotted blood and retroperitoneal haematoma



II- Fatal type

- The tear is deep or the pedicle is ruptured and hemorrhage is so massive that rapid death occurs.
- Small vessels in the spleen and sinusoidal lack muscle coats so do not constrict to stop bleeding.

III- Delayed rupture

- The initial shock is followed by a long lucid interval (may extend to a few days or weeks) after which patient presents with internal hemorrhage.
- This delay of clinical presentation may be due to
 - a) Subcapsular haematoma that gradually enlarges and rupture
 - b) Omentum seals perisplenic hematoma then later retracts
 - c) A clot stops bleeding but is later dislodged when the blood pressure rises or digested by enzymes from an injured pancreas.

3 stages may be present:

- 1) Stage of shock
- 2) Stage of recovery (lucid interval) due to arrest of bleeding from hypotension
- 3) Stage of internal hemorrhage due to rise of blood pressure after resuscitation

- If the patient is in severe shock, there is no need for investigations. The surgeon depends on clinical findings & proceeds for immediate laparotomy to stop bleeding

Investigations

I- Radiological

1) Abdominal Ultrasound or CT scan (most useful)

- Diagnostic
- Accuracy > 90%.
- Serial examinations to monitor the haematoma size.

2) Plain x-ray

- Poor diagnostic
- May reveal fracture of one or more of lower ribs

II. Laboratory

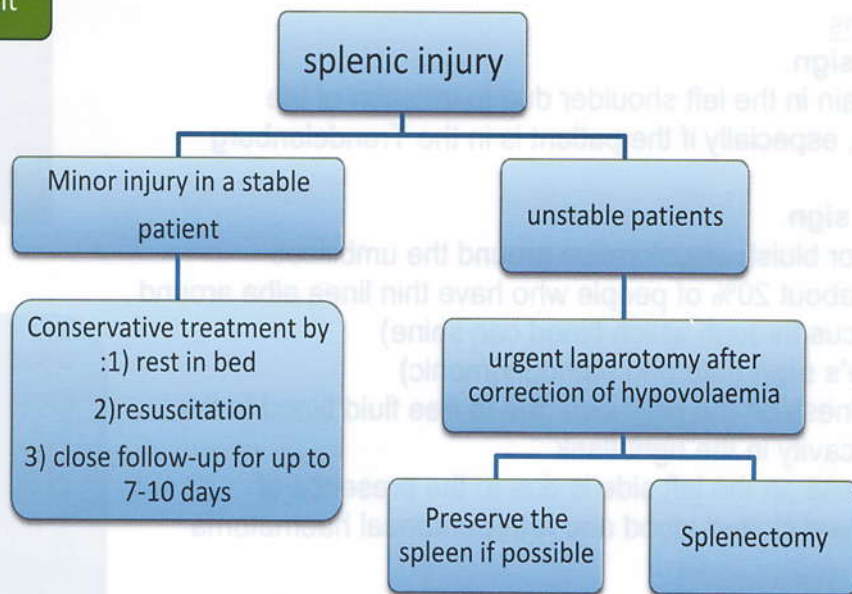
Repeated blood picture →

Declining hemoglobin and haematocrit denote hemorrhage.

III. Instrumental

Peritoneal lavage → reveals blood

Treatment



-In children <15 years every attempt should be made to preserve the spleen due to the vital role in the immune mechanism in this age.

Spleen preservation can be done either by:

- 1) Suture of a small laceration.
- 2) Partial splenectomy.
- 3) Compression of a lacerated spleen in a polyglactin (vicryl) mesh.

-**Splenectomy** is mandatory if there are:

- a) Deep lacerations
- b) Avulsion of the splenic pedicle.

-Post splenectomy (with 2 weeks) triple vaccination against:

Pneumococci, meningococci and H influenzae type B guards against severe infections

- In children → repeated every 5 years until the age of 18.
- For adults → antibody titres every 5 years determine the need for revaccination

After conservative surgery, meticulous observation is necessary as the patient may rebleed.

Functions of the spleen

1. Fetal erythropoiesis

- From the 5th to 8th month of fetal life
- This function does not continue in the normal adult.
- If the bone marrow production of red cells becomes defective after birth as in myelofibrosis → splenic erythropoiesis resume

2. Sequestration of old blood cells.

- Aged and abnormal erythrocytes, abnormal granulocytes, normal and abnormal platelets and cellular debris are cleared by spleen.
- at normal conditions, platelets survive 10 days circulation.
- 1/3 of the normal platelet pool is sequestered in the spleen.
- In splenomegaly, up to 80% of platelets are sequestered in the spleen → may lead to thrombocytopenia.

3. Defensive functions

- Macrophages phagocytose foreign substances as: bacteria, fungi, protozoa and particulate matter.
- plays an important role in proliferation of T and B lymphocytes.
- It helps in the production of various antibodies:
 - a) IgM.
 - B) Tuftsin → stimulates phagocytosis by neutrophils.
 - c) Opsonin → binds to bacteria and fungi to make them more susceptible to phagocytosis.
 - d) Properdin → fixes complement to bacterial and fungal surface prior to phagocytosis.
 - e) Interferon → is an antiviral antibody

Infections of the spleen

Acute abscess

Incidence : a rare condition occurring

Etiology : a) Specific fever, e.g. typhoid.

b) Blood borne from a distant septic lesion or during systemic pyaemia.

Treatment: Percutaneous drainage under ultrasound or CT guidance

Splenectomy may be very difficult due to dense adhesions

Schistosomiasis of the spleen

Etiology

Infection with: a) *Schistosoma mansoni* in 75%

b) *Schistosoma haematobium* in 25% of cases

Complications

- 1) Susceptibility to injury.
- 2) Susceptibility to infarction.
- 3) Hypersplenism.

Clinical picture

Manifestations of Schistosomal splenomegaly:

- 1) Dragging pain, if it is very large.
- 2) If splenic infarction occurs → pain due to peritoneal or diaphragmatic irritation.
- 3) Splenic enlargement may cause secondary hypersplenism

Treatment

Splenectomy rarely needed, only in:

- a) Hypersplenism.
- b) Huge splenomegaly which is burden to the patient
- c) Recurrent attacks of pain secondary to splenic infarctions

Hydatid disease

May lead to the formation of single or multiple cysts of the spleen

Treatment is splenectomy.

Malarial spleen

Incidence: common in endemic areas.

Gross picture: spleen may reach a huge size and is soft.

It acts as a reservoir for malaria parasites and, every now and then, causes an acute attack.

Complications: Malarial spleen is very liable to rupture as it is soft

- Causes of splenic enlargement in Schistosomiasis

1. In early stages results from reticuloendothelial hyperplasia due to deposition of ova in the splenic radicles.
2. With the advent of hepatic fibrosis → chronic venous congestion → progressive enlargement of the spleen (congestive splenomegaly)

Gross picture

1. The spleen is enlarged and has a thick capsule.
2. Perisplenitis may result in adhesions to the diaphragm.
3. The splenic vessels are markedly enlarged & tortuous

- Most of the complaints in patients with Schistosomal hepatosplenomegaly are 2nd to portal hypertension.
- Unlike liver cirrhosis, impairment of liver functions with Schistosomiasis is a minor

splenomegaly

Causes of splenomegaly

1) Infections

- a) **Bacterial:** Typhoid and paratyphoid, tuberculosis, abscess & syphilis.
- b) **Viral:** Infective mononucleosis and psittacosis.
- c) **Parasitic** → Schistosomiasis.
- d) **Protozoal** → Malaria

2) Blood diseases

- a) Hemolytic anemia
- b) Leukemia
- c) Thrombocytopenia
- d) Myelofibrosis.
- e) Polycythaemia Vera

3) **Metabolic** causes as Gaucher's disease and porphyria.

4) **Collagen diseases:** Felty's syndrome and Still's disease.

5) Portal hypertension

6) **Cysts of the spleen**, e.g., hydatid and blood cysts.

7) **Tumours** of the spleen



Causes of huge splenomegaly

- a) Thalassemia
- b) Gaucher's
- c) Myelofibrosis.
- d) Malaria.
- e) Portal hypertension.

Clinical picture

- An intra-abdominal swelling in the left hypochondrium (may descend downwards medially in the direction of the umbilical region).
- With inspiration → moves downwards and medially
- It has a sharp medial border (a notch commonly felt)
- The surface is usually smooth (nodular in lymphoma)
- The fingers cannot be insinuated between the spleen and the costal margin so the upper border cannot be reached.
- To differentiate from renal swelling → a splenic swelling does not fill the loin and does not ballot.
- Dull on percussion

Hemolytic anaemias of surgical interest

1) Hemolytic anaemias that are treated by splenectomy

- a) Hereditary spherocytosis is cured by splenectomy.
- b) Some cases of Thalassemia (less beneficial than in spherocytosis)
- c) Sickle cell anemia or the autoimmune type (rarely)

2) Sickle cell anemia

- Patients develop acute attacks if exposed to hypoxia.
- If they require surgery for any reason → the anaesthetist should ensure adequate oxygenation all through the operation

Tumors of the spleen:

- Incidence: are not common.

They may be:

a) Part of generalized reticuloendothelial affection as in lymphoma and leukemia.

b) A tumour that is confined to the spleen:

1. Benign tumours as lymphangioma and cavernous haemangioma.

2. Localized forms of lymphoma.

3. Secondary tumours are rare.

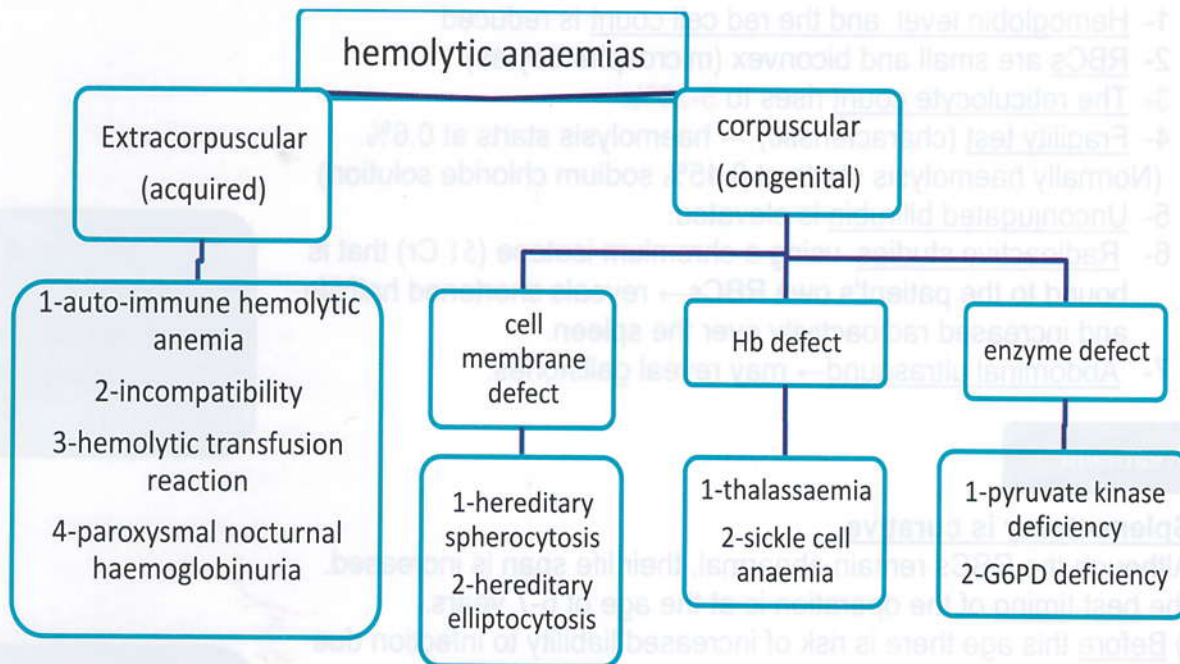
(May be infiltrated by direct extension from carcinoma of the stomach or pancreas)

- The normal spleen cannot be palpated clinically.

- If the spleen is palpable under the costal margin, it is enlarged at least 3 times its size.

- Enlargement of the spleen may reach below the level of the umbilicus or even down to the right iliac fossa

Hemolytic anaemias



Hereditary spherocytosis

Pathology

Inheritance: autosomal dominant (males = females)

Pathogenesis:

- A defect in the red cell membrane due to deficiency of certain proteins → the cells being smaller, less deformable and spherical instead of being biconvex discs.
- The spleen recognizes these blood cells as abnormal & destroys them.

Complications

- 1- Gallstones (Pigment stones in up to 60% of patients.) They may occur under the age of 10.
- 2- chronic leg ulcers may develop (rare)

Clinical picture

- Type of patient: at early infancy or may be delayed.
- General: There is mild hemolytic jaundice accompanied by mild to moderate anemia.
- Local: splenomegaly and hepatomegaly
- hemolytic crisis
- **Precipitated by**: infection
- Presents with**: severe pallor, fever, abdominal pain & vomiting followed by jaundice

-In hemolytic disorders the reduced red blood cell survival is demonstrated by measuring the disappearance of the patient's radioactive red blood cells (labeled with ^{51}Cr).
-The splenic role may be evaluated by determining the relative uptake of radioactivity by spleen & liver.

Investigations

- 1- Hemoglobin level and the red cell count is reduced
- 2- RBCs are small and biconvex (microspherocytes).
- 3- The reticulocyte count rises to **5-20%**.
- 4- Fragility test (characteristic) → haemolysis starts at 0.6%.
(Normally haemolysis starts at 0.45% sodium chloride solution)
- 5- Unconjugated bilirubin is elevated.
- 6- Radioactive studies using a chromium isotope (^{51}Cr) that is bound to the patient's own RBCs → reveals shortened half life and increased radioactivity over the spleen.
- 7- Abdominal ultrasound → may reveal gallstones.

If the patient have gallstones, cholecystectomy is simultaneously performed

Treatment

- Splenectomy is curative.

- Although the RBCs remain abnormal, their life span is increased.
- The best timing of the operation is at the age of 6-7 years.
 - a) Before this age there is risk of increased liability to infection due to the loss of the immune mechanism of the spleen.
 - b) After this age, the chance of developing gallstones is high.
- During surgery, search for accessory spleens (spleniculi) is important to avoid recurrence of hemolytic attacks.

Immune thrombocytopenic purpura (ITP)

Etiology

- Was formerly known as idiopathic thrombocytopenic purpura
- It is now known to be an autoimmune disease
- Platelets are sensitized with an autoantibody → early sequestration by the reticuloendothelial cells (mainly the spleen and liver)

Clinical picture

- Type of patient: most commonly in children and young adult females

- The course: intermittent (in remissions and exacerbations)

- Symptoms:

- a) Attack of bleeding under the skin (petechiae & ecchymosis) or mucous membranes, or from an orifice, as the nose, urinary or gastrointestinal tract.
- b) Menorrhagia (common in affected young females)

- Signs:

- a) Pallor
- b) Slight enlargement of the spleen
- c) Intracranial hemorrhage (rare but the most frequent cause of death)

- Pathology:

1. Low platelet count.
 2. Bone marrow contains normal or increased megakaryocytic.
 3. No systemic disease or intake of drugs that cause thrombocytopenia.
 4. The spleen is either a source of antibody production or as a major destruction site for sensitized platelets
- A huge spleen is against the diagnosis of ITP

Investigations

1. Platelets number decreased(may drop to 30 000/uL)
2. Red and white blood counts mildly diminished
(Due to repeated hemorrhages)
- 3- Bleeding time is prolonged up to 20 min (normally 2-5 min)
- 4- Coagulation time is normal.
- 5- Bone marrow contains a large number of megakaryocytes, with failure of budding.

Treatment

- **In children:** short courses of corticosteroids or immunoglobulins are usually followed by recovery.

- **In adults:** the disease relapses are frequent.

- Splenectomy indications:

- a) Persistent need for intake of corticosteroids for more than 6 months to maintain platelets count about 30,000/uL
- b) Marked side effects of corticosteroids

Hypersplenism

Definition

- The spleen is overdoing its function of getting rid of old blood Cells → destroys also young healthy blood cells → pancytopenia
- The bone marrow becomes hyperactive to compensate for the low blood cell counts.

Clinical picture

- 1) frequent infections (due to low WBCs)
- 2) petechiae and ecchymosis (due to low platelet count)
- 3) pallor and easy fatigue (due to anemia due to low RBCs)

Investigations

- 1) Blood picture reveals low counts of RBCs, leucocytes, and platelets with elevated reticulocyte count.
(One type of blood cells may be affected more than others)
- 2) Peripheral blood smear is devoid of any diagnostic features of leukemia or myeloproliferative disease.
- 3) Bone marrow reveals pancellular hyperplasia.
- 4) Confirmation is by injecting the patient's own RBCs after being tagged with radioactive chromium (51Cr) and estimating the half-life of RBCs → Patients with hypersplenism show diminished half-life of RBCs and elevated spleen-liver radioactivity ratio.

Treatment

Splenectomy

-Differential diagnosis of thrombocytopenia

1. Diminished production by the bone marrow in patients with aplastic anemia or 2ry to cancer chemotherapy.
2. Increased platelet consumption in DIC
3. Increased platelet destruction by the spleen as in:
 - a) ITP
 - b) 2ry to drug intake or certain infections
 - c) Hypersplenism

- Good response to corticosteroids predicts a good response to splenectomy

-Types of hypersplenism:

1) Primary hypersplenism (is the idiopathic type)

- The etiology not determined but 51Cr tagged red cell sequestration studies indicate that the spleen is the major site for phagocytosis.

2) Secondary hypersplenism

- Pancytopenia may arise from portal hypertension
- Portal hypertension → Splenomegaly with vascular congestion → destruction of the circulating cells within the spleen.

Splenectomy

Indications

- 1) Injury of the spleen
- 2) Hematological diseases:
 - a) Hereditary spherocytosis.
 - b) Immune thrombocytopenic purpura (ITP).
 - c) Some patients with acquired hemolytic anaemias.
 - d) Some patients with Thalassaemia who require frequent blood transfusions, particularly if they have acquired hemolytic antibodies.
 - e) Few patients with sickle cell anemia
- 3) Lymphomas.

Splenectomy performed to establish diagnosis and to reduce the dose of radio or chemotherapy.
- 4) During radical surgery for: stomach, esophagus or pancreas.
- 5) Hypersplenism
- 6) Splenic cysts
- 7) Splenic abscess

Complications

Complications that are common to all operations

- 1) Reactionary hemorrhage if a ligature slips off an artery or a vein
- 2) Atelectasis and pneumonia as upper abdominal incisions are painful and restrict depth of respiration.
- 3) Deep vein thrombosis from hypercoagulability that follow splenectomy due to rise in platelet count.
Thrombocytosis usually resolves within a month.
- 4) Surgical site infection.

Specific complications of splenectomy

- 1) Acute gastric dilatation
- 2) Portal vein thrombosis dt post-splenectomy thrombocytosis
- 3) Pancreatic fistula may follow damage to the tail of the pancreas during mobilization of the splenic pedicle → may be accompanied by left pleural effusion, peritoneal effusion, or abdominal wound dehiscence.
- 4) Subphrenic haematoma or abscess
- 5) Post-splenectomy bacterial infections especially the encapsulated types (Streptococcus pneumonia, Neisseria meningitides and Haemophilus influenzae type B)
 - Risk is higher if splenectomy is done in childhood.
 - A serious form is overwhelming postsplenectomy infection (OPSI)

-OPSI is fatal so splenectomized persons should receive vaccines against Pneumococci (pneumovax), meningococci and Haemophilus influenzae type B.

- For elective splenectomy, the vaccines are taken 2 weeks before surgery.
- For emergency splenectomies, they are taken 2 weeks postoperatively.
- In children, vaccinations are repeated every 5 years until the age of 18.
- For adults who had splenectomy and were vaccinated, antibody titres every 5 years determines need for revaccination
- Patients are instructed that at first sign of febrile illness antibiotics should be taken

The intestine

Functions of the small intestine

1. Absorption (**main function**) → Fat, protein, CHO, vitamins, H₂O & electrolytes
 - With few exceptions (e.g. iron and calcium), the small intestine absorbs regardless body composition.
 - vitamin B12 to be absorbed → combines with the intrinsic factor secreted by the stomach → absorbed in the distal ileum
2. Important secretory and digestive functions through the succus entericus (intestinal juice)

Functions of the colon and rectum

1. Right side of the colon → absorbs H₂O & Na → concentrate waste products.
2. The left side of the colon → reservoir for solid faeces until time of defecation.
3. Mucus secreted by the mucosa acts as a lubricant. Potassium and bicarbonate are also secreted in small quantities.

Principles of colon surgery

Colon surgery needs special consideration because its anastomosis is more liable to disruption, leakage and peritonitis than small bowel due to:

1. The high content of both aerobic and anaerobic organisms.
2. Constant gaseous distension.
3. Incomplete serous coat.
4. The terminal arteries are poorly connected with each other.

Preoperative preparation for elective surgery

- 1- Patient counseling (particularly for undergoing intestinal stoma)
- 2- Improving nutritional status.
- 3- Bowel preparation

-If the large bowel is empty and bacterial flora is reduced at time of resection → reduced risk of anastomosis leakage and wound sepsis
-In elective cases where the colon is well prepared (is empty and clean) → 1st anastomosis (in the same session as the resection) can be performed. There are two methods of bowel preparation.

a- Mechanical preparation

• Standard preparation

- i) Non residue diet for 4 days before surgery.
- ii) Enemas and mild laxatives for 2-3 days before the operation.

• Rapid preparation

- An alternative that can be done one day before surgery by either:
- i) Whole gut irrigation using 2-4L/hour of a balanced crystalloid solution passed via a nasogastric tube until the patient passes clear fluid per rectum.
 - ii) Mannitol.
- One liter of flavored mannitol is given orally or by a NG tube.

-Resection of a long segment of the distal small intestine produces vitamin deficiency.

-Ingested fluid and salivary, gastric, biliary, pancreatic and intestinal secretions present a total of 5-9 L/day but only 1-2 L pass the ileocaecal valve to colon.

-Excessive diarrhoea → K⁺ & HCO₃ loss → metabolic acidosis

- Whole gut irrigation is not used in patients with known cardiac or renal disease and in those with partially obstructed colon

-Metchlopramide can be used to inhibit vomiting induced by mannitol

b- Chemical preparation.

- Intestinal antiseptics (taken orally to reduce the colon bacteria)
- Neomycin and metronidazole (Flagyl) for 2 days
- Will cover the Gram -ve bacilli and anaerobes (normally resident in the large intestine)
- 4- Perioperative prophylactic parenteral antibiotics
 - To minimize septic complications following
 - Systemic antibiotics are administered immediately before surgery and are continued postoperatively for one day.
 - A combination of a cephalosporin or an aminoglycoside with either metronidazole or clindamycin

- For a successful anastomosis → the two bowel ends should be adequately vascularized and sutured without tension

Operative procedure

I- Resection

- The extent of resection is controlled by the arterial blood supply and by the disease process.
- In radical surgery for malignant tumors → remove the draining lymphatics and LNs
- Division of the peritoneal attachments allows adequate mobilization of the bowel on its mesentery.

II- Anastomosis, done by either:

1. Hand suturing

- Is commonly done in 2 layers of interrupted sutures →
 - i) The 1st layer → includes the whole wall thickness
 - ii) The 2nd layer → incorporates the serosa and muscle only (to invert and seal the suture line)
- Some surgeons prefer a single layer of interrupted sutures.
- The defect in the mesentery is closed to prevent an internal hernia.

2. Mechanical staplers are increasingly used.

They are faster but more costly.

Emergency surgery

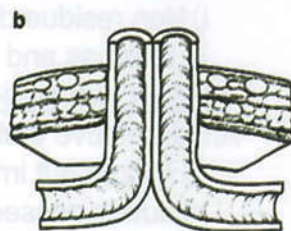
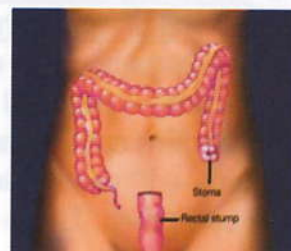
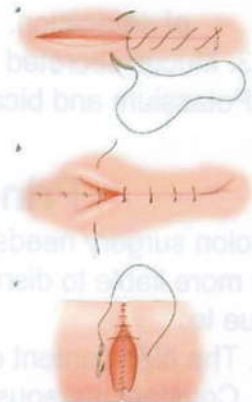
- Indications:

IO, perforation, toxic dilatation or massive bleeding from the colon

- According to general condition of patient

- I- **In bad general conditions** → a temporary proximal colostomy done → later on resection and anastomosis when suitable
- II- **In good general condition** → resection of the diseased colon then anastomosis decision depend on the site →
 - a) **In right colon** → 1^{ry} anastomosis (ileo-transverse anastomosis = ileum to transverse colon)
 - b) **Other parts of the colon** → primary anastomosis are avoided because of the high possibility of disrupted suture line and leakage. The options are:
 - 1- **Hartmann's procedure**
 - Proximal end is opened to the skin as a temporary colostomy
 - Distal end is closed by sutures and replaced in the abdomen
 - 2- **Mikulicz method** → Both ends are opened to the skin
 - Proximal one → temporary colostomy
 - Distal one → as a mucus fistula.

Both Hartmann's and Mikulicz are followed by 2nd operation to restore bowel continuity within a few weeks after proper bowel preparation.



Ileostomy

Indications

Proctocolectomy for ulcerative colitis or familial polyposis coli

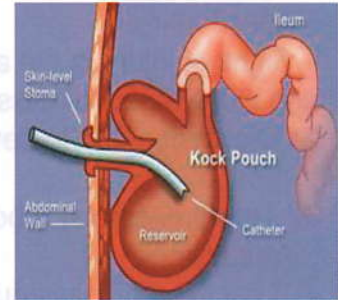
Types

1-Conventional ileostomy

- Ileum protrudes from the skin as a stoma facilitating direct delivery of the irritant small bowel content into an appliance.
- Are incontinent.

2- Continent ileostomy

- Done by fashioning a valve with an underlying reservoir (**Kock pouch**) which the patient regularly evacuates by passing a tube.



Colostomy

See operative book

Intestinal trauma

Etiology

1. Blunt abdominal trauma

- As in road traffic accidents (RTA) or blows to the abdomen
- Presence of seat belt injuries → direct attention to the possibility of intestinal injury.

2. Penetrating trauma as stabs and bullets.

- High velocity missiles are more damaging than low velocity ones and stabs.

3. Indirect trauma as blast injuries are known to affect the colon.

4. Iatrogenic injury (is increasing), examples include:

- a. The **duodenum** → during endoscopic sphincterotomy done to remove a stone from the CBD
- b. The **colon** → during colonoscopic diathermy excision of polyps.
- c. The **intestine** → during laparoscopic surgery when the pneumoperitoneum needle is introduced in an area where the intestine is adherent to the anterior abdominal wall.

- Colon injuries are more dangerous than those of the small intestine because of the magnitude of intra-abdominal contamination

- Types of injury:

1. Contusion and haematoma.
2. Rupture (Complete or incomplete and single or multiple)
3. Tears of mesentery or mesenteric vessels → hematomas and gangrene

Sequelae

1. Peritonitis due to the escape of intestinal contents
2. Internal haemorrhage (is less important than peritonitis)
3. Hypovolaemia and septic shock.
4. Paralytic ileus.
5. Intestinal obstruction due to healing of injuries by strictures and massive adhesions

Clinical picture

Symptoms

- 1-History of trauma
- 2-Abdominal pain (starts at the site of injury then spreads)

Signs

I- General signs: Tachycardia, fever, and hypotension.

II- Local signs :

-Inspection

1- Signs of injury in the abdominal wall → as bruises or the inlet and exit wounds of missiles.

2- Distension due to developing ileus.

-Palpation

3- Tenderness and rebound tenderness.

-Percussion

4- Lost liver dullness (due to free air in the peritoneum)

5- Shifting dullness (due to free intraperitoneal fluid).

Investigations

1. Laboratory tests. Leucocytosis and hemodilution

2. Radiology

a) Plain X-ray of the abdomen may show:

i- Free air under the diaphragm.

ii- Multiple fluid levels due to ileus.

iii- Bullets and sharpnells.

iv- Fractures .

b) Ultrasound or CT scan

-may reveal a haematoma or intraperitoneal collection.

3. Diagnostic peritoneal lavage (DPL)

May show free blood, bile or intestinal contents

Treatment

Priorities of multiple trauma management should be followed

I- Preoperative preparation

1- Anti-shock measures.

2- Antibiotics (that cover Gram -ve bacilli and anaerobes → combination of 3rd generation cephalosporin & metronidazole)

3- Tetanus toxoid boosting dose

4- Insertion of NG tube & a self retaining urethral catheter.

II- Operation (abdominal exploration = laparotomy)

1- Midline abdominal incision,

2- Full abdominal exploration for solid and hollow organs

3- Bleeding should be urgently stopped.

4- Leaking intestine is dealt with according to the site

A-Small intestinal and right colon injuries: according to the type

i) **Tidy sharp injuries** → sutured in two layers.

ii) **Ragged injuries** → trimming of the edges and suturing

iii) **ischemic or gangrenous** segments (due to mesenteric vessels injury, extensively contused segments and for multiple close tears) → Intestinal resection & restoration of intestinal continuity by 1st anastomosis

- The frank picture of peritonitis may be delayed in patients with a tiny perforation.

Sometimes, the injured segment is contused & it takes some time to perforate.

- Whenever in doubt, it is better to keep the patient under observation in the hospital for 24 hours

- The diagnosis of bowel injury is mainly clinical (Investigations done only in doubtful cases)

- All intestinal injuries require laparotomy



The peritoneal cavity is irrigated with Copious amounts of saline before abdominal closure

B-Transverse and left colon injuries : according to type

- 1- Localized injuries that can be exteriorized → the injured part is brought out to the skin through an opening in the anterior abdominal wall → acts as a colostomy.
- 2- If the injured part cannot be exteriorized → the tear is sutured and protected by proximal colostomy to divert the faeces away.
- 3- In cases where resection of a segment is done →
 - i- The proximal end is brought out as terminal colostomy.
 - ii- The distal end is either brought out as a mucus fistula (Mikulicz method) or is closed (Hartmann's procedure).
 - iii- Elective anastomosis done after 3 weeks.

Colostomy is electively closed after 3 weeks with adequate colon preparation

Intestinal fistulae

Definition

An abnormal communication between 2 epithelialized surfaces, is usually formed by granulation tissue but may sometimes be lined by epithelium

Etiology

I-Congenital:

As a patent vitello-intestinal duct that discharges at the umbilicus.

II- Acquired:

1- Trauma :

- After an abdominal operation (**80 %**) due to :
 - a. Unrecognized intestinal injury.
 - b. Failure of an intestinal anastomosis. Caused by →
 - i. Poor vascularity.
 - ii. Anastomosis under tension.
 - iii. Anastomosis in the presence of sepsis.
 - iv. Distal obstruction.
 - v. Lack of proper surgical technique.
 - vi. Presence of a specific pathology as Crohn's disease.
 - vii. Generalized diseases that impair healing as hypoproteinaemia.
- Abdominal trauma as in RTA

2- Inflammation as:

a- Colonic diverticulitis b- Crohn's disease c- Radiation enteritis

3- Tumours

Complications

1. Metabolic effects

- include:

- a. Dehydration
 - b. Malnutrition as Hypoalbuminaemia.
 - c. Electrolyte disturbances as hyponatraemia and hypokalaemia.
 - d. Acid-base disturbance → causes acidosis.
- Common with high output fistulae
 - Are the results of malabsorption and loss of intestinal content
 - Sepsis → leads to severe catabolism → aggravates malnutrition

Classification:

-According to site of opening:

1. Internal fistula

- connect the intestine to hollow viscera (GIT, UT or vagina)

2. External fistula

- Connects intestine to the skin.

- Are further classified into→

- a) low-output fistulae → discharge < 500 ml/day
- b) high-output fistulae → discharge > 500 ml/day. The

II- According to origin in intestine

(nown by the nature of discharge)

1- Bile-stained discharge → from the duodenum or jejunum.

2- Fluid faecal discharge → from the ileum or caecum.

3- Semisolid faecal discharge → from the distal colon

2. Sepsis (a major problem)

- If the fistula track is not effectively walled off → enteric contents escape → intraperitoneal abscess.

3. Skin irritation and maceration

Due to continuous flow of intestinal contents

Investigation

- The aim is to detect :
 - 1- The level of the fistula
 - 2- The presence of bowel disease
 - 3- The presence of distal obstruction
 - 4- The presence of intraperitoneal sepsis.
- The means are:
 1. Clinical assessment.
 2. Barium meal follow-through.
 3. Fistulography
 4. Ultrasound or CT of abdomen to detect abscess cavities.

Management

- Managing an intestinal fistula proceeds in steps:

1. Resuscitation and skin protection

- By IV fluids (to restore normal blood volume), fluid, and electrolyte balance.
- Blood transfusion may be needed.

2- Skin protection

- Start as early as the fistula is recognized, if excoriation occurs → difficult management.
- An adhesive cover and a disposable collection bag are applied to the skin around the fistula → the skin is protected and the effluent can be measured.

3- Nutritional support

- a. With high output or proximal fistulae → Parenteral nutrition
- b. With low output and distal fistulae → Enteral nutrition

4. Definitive treatment

A- External fistula:

- i) If the output is decreasing → Continue conservative treatment
- ii) Surgical intervention is indicated for:
 - Cases that show no improvement over 4-6 weeks.
 - Distal obstruction.
 - Active disease at the fistula site as Crohn's disease or malignancy.
 - Total discontinuity of the bowel ends.
 - Mucocutaneous continuity.

B- Internal fistula.

- Spontaneous closure is rare and many do not require correction.
- Surgery is indicated for:
 - i) Fistula that bypasses a long segment of intestine → malabsorption
 - ii) Internal fistulae to the urinary tract



- The amount of fistula output has an important bearing on its metabolic effects.
- An abscess should be drained either surgically or is aspirated under Sonographic or CT guidance
- The majority of intestinal fistulae heal spontaneously if:
 - 1- Sepsis eradicated
 - 2- nutritional status is maintained
 - 3- Distal obstruction, is relieved (if present)
- Surgery is in the form of resection of the affected segment & anastomosis of healthy ends
- A good nutritional status allows resection of the diseased bowel segment & anastomosis in one stage

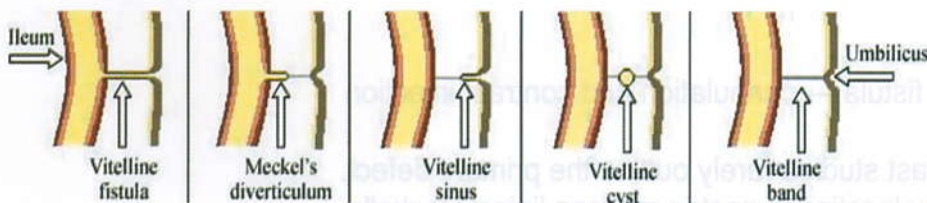
Meckel's diverticulum

Etiology

- Caused by persistent patency of the proximal part of the vitello-intestinal (omphalo-mesenteric) duct of the embryo

The abnormal persistence of the duct may produce other anomalies as well:

1. Persistent patency of the whole duct → faecal fistula connecting the ileum with the umbilicus
2. persistent patency of the middle part of the duct → enterocyst
3. Persistent umbilical extremity of the duct → everted producing an umbilical polyp
4. A fibrous band remaining after obliteration of the duct



Intestinal diverticula

-Definition:

A blind pouch that is continuous with the lumen of a hollow viscus (gut or urinary bladder)

-Site:

- Occur anywhere in the bowel from the duodenum to sigmoid colon.
- The commonest are Meckel's diverticulum & diverticula of the colon

Pathology

-Is a true diverticulum → consists of all layers of the bowel wall.

-Site → lies on the antimesenteric border

- Has separate blood supply via a branch of the SMA.

-It may contain ectopic gastric, pancreatic and colonic cells.

- Rule of 2-3 , As an approximation:

- 1) Occurs in **2-3%** of the population
- 2) It is situated in the ileum **2-3** feet from the caecum
- 3) **2-3** inches in length.
- 4) Complications occur in about **2-3%** of affected people.
- 5) Males: female ratio = **2:1**
- 6) **2-3 cm** in diameter



Rule of 2 in Meckel's

Clinical picture

1. May be accidentally discovered at operation for another pathology
2. May present by clinical picture of its complication as:
 - Intestinal obstruction.
 - Painless rectal bleeding in childhood.
 - Acute abdominal pain simulating appendicitis.

- A Meckel's diverticulum is the most prevalent congenital anomaly of GIT

-Clinically it is impossible to differentiate appendicitis from Meckel's diverticulitis → usually explored on the provisional diagnosis of acute appendicitis.

- If appendix found normal at operation → look for a Meckel's diverticulum.

Complications

1. Intestinal obstruction (the commonest complication)

-It may be due to:

a) Intussusception:

The ectopic mucosa at the base of the diverticulum acts as a foreign body and forms the apex of an ileo-ileal intussusception.

b) Fibrous band that:

i- Attach the diverticulum to the umbilicus → Pressure on an intestinal loop

ii- Allow rotation of the ileum around its axis → **volvulus**

2. Peptic ulceration and bleeding (particularly in childhood)

-Caused by the ectopic gastric mucosa

-The ulceration occurs in the intestinal mucosa adjacent to the base of the diverticulum.

3. Acute diverticulitis

-Is similar to acute appendicitis.

-Gangrene and perforation may occur → peritonitis

4. Littre's hernia.

Become content of an inguinal or a femoral hernia

Bleeding 2nd to a Meckel's diverticulum is the commonest cause of lower GIT bleeding in children



Investigations

1- For umbilical sinus or fistula → cannulation and contrast injection via the umbilical tract.

2-Upper and lower contrast studies rarely outline the primary defect.

-Technetium Tc 99m may localize in gastric mucosa lining Meckel's diverticulum and may identify the source of hematochezia or melena.

-Retention of dye in the mucous and parietal cells is enhanced by giving cimetidine, **30 mg/kg IV, 30 min.** before administration of the radiotracer nuclide

Treatment

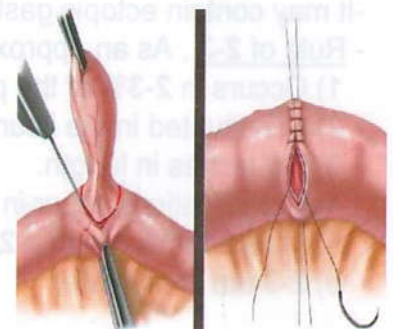
I-Symptomatic cases

Resection is indicated.

II-Accidentally discovered at laparotomy

1-in children & young adults and in those with an attached band → Resection is indicated

2- In other instances (particularly in patients >40 years) → leave it as risks of resection outweigh the advantages.

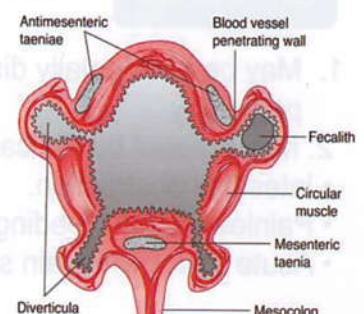


Diverticular disease of the colon

Etiology

- Lack of fibre in the diet → chronic constipation and increased intraluminal colonic pressure.

- Increased muscle spasm and segmentation may play a role.



1- Nature

- Represents pulsion diverticula of the colonic mucosa

2-Site (any area of the colon may be involved)

-The sigmoid colon is the commonest site affected.

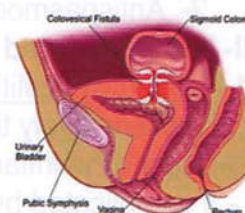
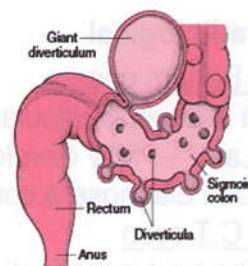
- The rectum is never affected.

3-Development.

-In the early stages there is only muscular spasm and incoordination.

-Next, the mucosa starts to bulge outwards through the circular muscular layer at the points of entry of blood vessels between the taenia at the anti mesenteric border

-This called diverticulosis or non complicated diverticular disease



Complications

1. Acute diverticulitis

- Is inflammation of 1 or more diverticula 2^{ry} to obstruction of its neck

2. Perforation → lead to localized or generalized peritonitis

- May be a sequel of acute diverticulitis.

3. Recurrent attacks of acute inflammation.

4. Bleeding

Condensed faeces → blood vessel erosion at neck of diverticulum

5. Colon stricture → lead to large bowel obstruction.

- 2^{ry} to chronic diverticulitis

6. Fistula formation.

A colovesical, colovaginal or colointestinal fistula

Clinical picture

Type of patient:

Uncommon before the age of 40, incidence increases with age.

I-Non complicated cases : either

1-the patient may be asymptomatic

2-complains of recurrent attacks of lower abdominal pain, distension and flatulence.

II-Complicated cases

1-Acute diverticulitis

- Mimics the clinical picture of appendicitis but on the left side.

- **Symptoms:** There is abdominal pain

-**Signs:** pyrexia with tenderness and guarding in LT iliac fossa.

-**Complications:** a pericolic abscess or generalized peritonitis.

2-Chronic diverticulitis

- A long history of recurrent attacks of pain with the passage of blood and mucous per rectum.

- On palpation there is a tender mass in the left iliac region.

3-Fresh bleeding per rectum.

- May be the first presentation of the disease.

- In profuse haemorrhage, diagnosis of the source of bleeding

Will be difficult by colonoscopy so do → Angiography (most useful)

-The mass of chronic diverticulitis has to be differentiated from carcinoma.
-In carcinoma the history is of a short duration and pain is absent (in 25%)

Investigations

I-radiological

1. Barium enema.

- In the prediverticular stage → a saw tooth appearance
- Later → fully developed diverticula visualized
- Can diagnose a concomitant lesion.

2. C.T scan

- The best investigation in acute diverticulitis
- It reveals thickening of the colonic wall, a peridiverticular abscess or extraluminal gas

3. Angiography

- The most useful in bleeding per rectum



II-instrumental

• Sigmoidoscopy

- Will detect the mouths of the diverticula
- It is of more value to diagnose any concomitant lesion.



Treatment

I-For uncomplicated cases (diverticulosis)

- 1- High fibre diet is prescribed.
- 2- Antispasmodics for the abdominal colics.

II- In complicated cases

1-Acute diverticulitis

- Conservatively the same as appendicular mass.
- Usually the inflammation settles on conservative treatment.
- If complicated by :
 - a) **Pericolic abscess** → open drainage or ultrasound guided percutaneous aspiration is performed.
 - b) **Generalized peritonitis** → urgent laparotomy → resect the perforated colon by a Hartmann's & do peritoneal toilet and drainage(the best)

Exteriorization of the perforated segment or suture of the perforation with a defunctioning colostomy is less successful

2. Chronic diverticulitis.

Colectomy is performed after adequate preparation of the colon.

3. Bleeding: a) Adequate resuscitation.

b) In majority of patients, the bleeding stops spontaneously.

c) If the bleeding is massive or persistent → do angiography to localize the site of bleeding and urgent Colectomy is performed.

-In acute diverticulitis → barium enema is contraindicated ,instead use CT scan

-Commonest causes of massive bleeding per rectum:

- 1-diverticular disease of colon
- 2- Angiodysplasia of colon

-Cancer colon is a commoner cause of bleeding per rectum but not massive bleeding

Inflammatory bowel disease

-The term "inflammatory bowel disease" is usually used to denote ulcerative colitis and Crohn's disease.

-However, bowel inflammation can be caused by other diseases, as:

- 1-Schistosomal colitis
- 2- Amoebic colitis
- 3- Ileocaecal tuberculosis
- 4- Typhoid enteritis

Ulcerative colitis

Definition

An inflammatory disease of the colon (colitis) characterized by ulcerations.

Etiology

- Is unknown.
- Immunologic, genetic & environmental factors are possible causes

Pathology

Site

- Begins at the dentate line of the anal canal and extends proximally.
- May involve rectum alone (proctitis) up to whole colon and rectum affection (pancolitis).

Macroscopic picture

- Extension is continuous with no skip areas of normal bowel
- Mucosal affection varies from granularity to extensive ulceration

Microscopic picture

- Only the mucosa and submucosa are involved (unlike crohn's)
- The characteristic histological feature is crypt abscesses formation in the depths of the glandular tubules with surrounding inflammation.
- The abscesses fuse to form ulcers
- Pseudopolyps are likely to be found

Complications

A- Intestinal

- 1-Toxic megacolon (3-5% of cases) → can be fatal.
 - 2- Haemorrhage
 - 3- Colon cancer
- The risk is higher in patients with pancolitis for >10 years.
 - Carcinoma on top of ulcerative colitis is often multicentric.

B- Extraintestinal

- 1-arthritis
- 2- uveitis
- 3-cholangitis
- 4- Liver cirrhosis
- 5- Skin lesions as: pyoderma gangrenosum & erythema nodosum.

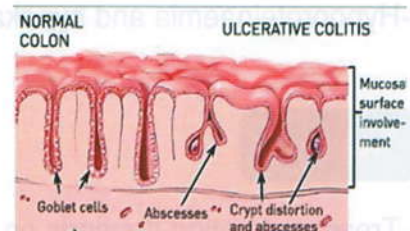
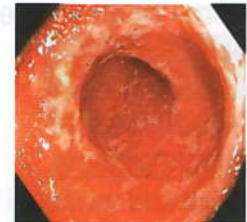
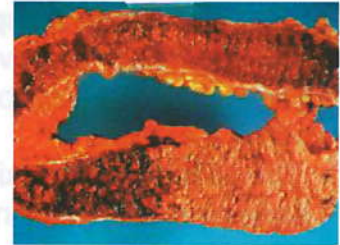
Clinical picture

Type of patient

- More common in females.
- The commonest age is the 3rd and 4th decade

The Complaint

- Watery diarrhoea mixed with blood, pus, mucus and tenesmus.
- Pain, fever, weight loss, and dehydration occur to varying degrees.



The course

- Characterized by the occurrence of remissions and relapses.

Clinical picture of complications

As Toxic megacolon (a very serious complication)

- There is severe diarrhoea with the passage of blood, mucous and pus in the stools.
- The patient is very toxic with severe pyrexia **39-39.5°C**.
- Abdominal examination → severe abdominal distension due to marked colonic atony.
- If not treated urgently → a fatal outcome.

Investigations

I-Radiological

Barium enema

- Reveals mucosal irregularity and ulcers
- Colonic shortening and loss of haustrations (pipe-stem appearance) in long standing disease.
- Contraindicated in the presence of toxic megacolon.

II-instrumental

Colonoscopy and biopsy

- Shows the mucosal abnormalities
- Detects the upper extent of the disease
- Is contraindicated in toxic megacolon

III-Laboratory

- CBC → Anaemia and leucocytosis (in acute cases)
- Hypoproteinaemia and hypokalaemia (in severe cases)



Treatment

- Treatment option depends on the severity of the disease.
- The disease is severe if there is ≥ 6 bloody motions/day with fever $> 37.5^{\circ}\text{C}$

1) Mild disease:

Oral 5-ASA or by enema

2) Moderate to severe disease:

- Oral 5-ASA or enema
- Prednisone oral or enema or IV hydrocortisone

3) Resistant cases:

- Immunosuppressive drugs as:
Cyclosporine or azathioprine or mycophenolate mofetil
- Biological agents as Infliximab

Aminosalicylic acid is a major drug in the treatment of ulcerative colitis → all patients with UC should take it

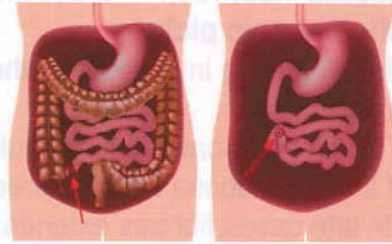
Nutritional therapy:

- In the majority of patients → a well balanced healthy diet
- Avoid only foods which are poorly tolerated (by experience)
- In SI strictures: avoid raw fruits & vegetables → precipitate I.O.
- In cases of combined proctitis & constipation → ↑ dietary fiber.

Surgical treatment

Indications

1. Failure of medical management to control the disease leading to a chronic ill health with persistent diarrhoea, anaemia and malnutrition.
2. Development of complications as toxic megacolon with perforation (urgent surgery) or colon cancer.
3. Long-standing pancolitis where biopsy shows dysplasia.
4. Stricture formation.



Surgical options

1. Panproctocolectomy (the standard treatment)

Excision of the whole colon and rectum with permanent ileostomy

2. Colectomy

-The colon is excised but the rectum may be spared and an ileo-rectal anastomosis is performed.

-Lifelong observation of the rectum is needed for fear of development of a rectal carcinoma.

3. Proctocolectomy with distal rectal mucosectomy.

-Excision of the colon & upper half of the rectum is performed.

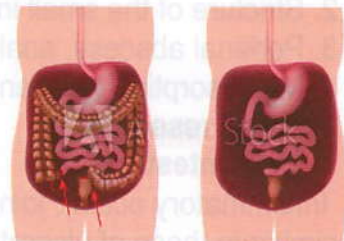
-The mucosa of the lower part of the rectum (the site of the disease) is cored out leaving the muscle wall.

- An ileal pouch is fashioned to act as a reservoir and is anastomosed to the anal canal within the preserved rectal muscles

- Not suitable in an emergency setting.

-Despite the risks, 95% of patients are satisfied with the procedure and have a good quality of life after IPAA

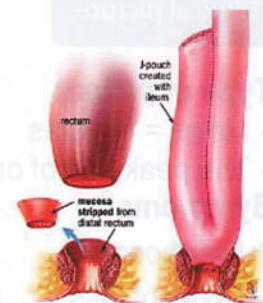
Colectomy with Ileorectostomy



Colon removed but rectum preserved

End of ileum attached to upper part of rectum

#173811640



Crohn's disease (regional ileitis)

Definition

An idiopathic chronic transmural granulomatous inflammation that can involve any area in the GIT

Etiology

- Unknown but may be:
- 1-Heridity
 - 2- Disturbances in the immune system.

Pathology

Site:

- Can affect any part of GIT from the mouth to the anal canal
- The commonest is the distal ileum → the old name "regional ileitis"
- Next common site is the colon followed by the anal canal.

Macroscopic picture

- Characterized by granuloma formation.
- The affected segment is thickened
- The mesentery is also thickened and contains enlarged LNs
- Characterized by skip lesions (healthy gut between the affected)



- In longstanding cases → multiple fissures occur with edematous mucosa inbetween → **a cobble stone appearance**

Microscopic picture

- Inflammation involves the whole thickness from mucosa to serosa (transmural)
- There is non-caseating granulomatous infiltration of the lymphatics of the submucosa with the presence of giant-cells.
- In late cases fibrosis extends into and obliterates the submucosa.



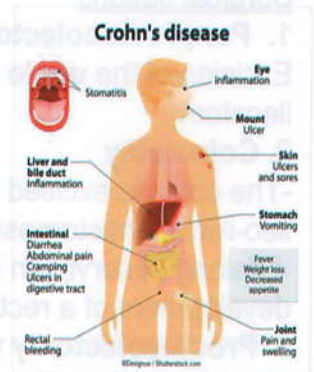
Complications

I- Intestinal

1. Abscess & fistula to another loop, bladder, vagina or to the skin.
2. Stricture of the small intestine → intestinal obstruction.
3. Perianal abscess, anal fistula and fissure.
4. Malabsorption in extensive disease or in those who had extensive intestinal resection.

II- Extraintestinal

Inflammatory ocular, joint, skin, and hepatobiliary lesions are similar to those of ulcerative colitis.



Clinical picture

Type of patient

- Males = females
- The peak age of onset is in the 2nd – 4th decades

Symptoms

- 1-Diarrhoea
 - 2-Abdominal pain
 - 3-wWeakness
 - 4- Weight loss
 - 5- Anorectal disease.
- 6-In old standing cases → may present with IO.

Signs

General → anaemia & malnutrition

Local → a mass in the right iliac fossa

Barium meal follow through is done for suspected SI disease while a barium enema is indicated for suspected colon disease

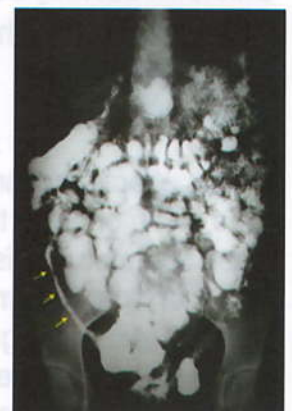
Investigations

I-radiological

- Barium meal and follow through : shows
- Segmental areas of stricture separated by **skip areas** of free bowel
- Cobble stone appearance of mucosa due to longitudinal fissures.
- It may also show internal or external fistulae.
- A narrowed terminal ileal segment is called **string sign of Kantor**

II-instrumental

- Colonoscopy and biopsy
- Of large bowel lesions proves the diagnosis
- Biopsy of suspected anal fissures and fistulae



Treatment

Medical treatment

The aim → to induce and then maintain a remission

1) For active small intestinal involvement:

-Prednisone PO or IV according to the severity and reducing the dose once improvement occur.

- For resistant cases → immunosuppressive drugs

2) For active colitis:

- In mild cases → sulphasalazine
- In moderate to severe cases → Prednisone
- In resistant cases → Immunosuppressive drugs

-Removal of long intestinal segments is avoided → leads to malabsorption
- Strictureplasty does not treat Crohn's Colitis, as there is a 7% risk of malignancy over 20 years. (Washington Manual of Surgery)

Surgery

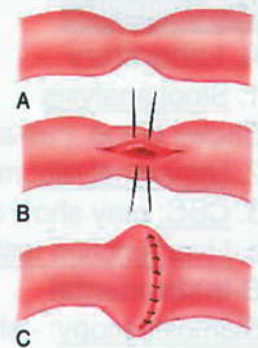
-Reserved for complications but becomes necessary in most cases.

- Indications :

- 1-Failure of medical treatment
- 2- Complications as: acute systemic sepsis , perforation , uncontrolled hemorrhage, failure to thrive and malnutrition
- 3- Dysplasia and malignancy.

Operations

- 1- Limited resection of the affected area as the patient may be subjected to further resections in the future,
 - 2- Stricuroplasty
- For localized strictures
 - Incise the stricture longitudinally & close it transversely → widen the lumen



Bilharzial colitis

Etiology

-Caused by infestation with *Schistosoma mansoni* and by *Schistosoma haematobium* (less commonly)

Pathology

Site → usually at the sigmoid colon and the rectum

-Chronic inflammatory cells surround the deposited ova → Bilharzial granulomas.

- The disease may produce:

- 1-Polyps
- 2- Ulcers
- 3- Bilharzial pericolic mass

- The condition is not precancerous and does not produce strictures.

Complications

1. Anaemia (due to chronic blood loss)
2. Hypoproteinaemia (due to chronic protein loss)
3. Partial rectal prolapse.
4. Prolapse and strangulation of a polyp

Multiple Bilharzial Polyps of large intestine



Clinical picture

Type of patient

The disease often affects young adult males in rural areas of Egypt.

Symptoms

1. Diarrhoea
2. Tenesmus
3. Weakness
4. colics
5. Passage of mucus and blood in stools.
6. Dyspepsia

Signs

General: Pallor & Clubbing of fingers (with ulcerated of polyps)

Local:

- 1-The sigmoid colon is palpably thickened and tender.
- 2- A hard nodular mass felt in the left iliac fossa (in the diffuse form)
The mass is mobile or fixed & is hard → simulate cancer (but no I.O.)

DRE: commonly detects multiple polyps.

Investigations

A. Laboratory

1. Stool analysis
Shows blood, pus and probably viable Bilharzial ova.
2. Urine analysis: may reveal associated urinary infestation.
3. CBC: may show anaemia.
- c. Liver function tests: may reveal hypoalbuminaemia.

B-Instrumental

- Sigmoidoscopy: - shows the mucosal changes.
- Examination of fresh biopsy or ulcer scrapings → Show the ova.

C- Radiological.

Barium enema

- May show multiple rounded filling defects of the polyps.
- The sigmoid colon may be pulled to the right iliac fossa



Treatment

- 1- Antibilharzial drugs → Oxamniquine or praziquantel
- 2- Polyps → polypectomy by fiberoptic colonoscopy

Surgical complications of typhoid and paratyphoid

A-Intestinal

1. Intestinal haemorrhage

- Due to ulceration of the affected Peyer's patches in the lower ileum. -The ulcer is parallel to the long axis of the gut.
- Occurs 2-3 weeks after the onset of the disease.
- Presents as dark blood per rectum (may be bright red if profuse)

2. Perforation of a typhoid ulcer

- May occur around 3rd week → peritonitis.
- Needs urgent exploration under cover of chloramphenicol
- Pus is evacuated
- The perforation is repaired or the affected ileal segment is resected

B- Biliary: 1-Acute non-calicular cholecystitis

2- Chronic cholecystitis (carrier state)

C- Vascular

DVT of the lower limbs due to prolonged recumbency

Intestinal tuberculosis

	Ulcerative tuberculous	Hyperplastic ileocaecal tuberculosis
Etiology	-2 nd to pulmonary TB -swallowing of TB bacilli	1 st infection due to ingestion of TB bacilli (bovine or human type) by patients with high resistance.
Pathology	-Present as multiple ulcers in the terminal ileum. -The long axis of TB ulcer lies transversely	-Site: ileocaecal region (commonest). - Intestinal wall is much thickened & lumen is narrow - Regional LNs early are early involved → may caseate.
Clinical picture	1.Diarrhoea 2.Abdominal pain 3. Weight loss	1.Abdominal pain 2. Intermittent diarrhoea 3.Mass in RT. iliac fossa in ill health pt.
Complications	1- Stricture → I.O. 2- Perforation (rare)	1- I.O. (if untreated) 2- Abscess & fistula (rare)
Investigations	<u>Barium meal follow through</u> → +ve sterlin sign (non visualization of terminal ileum and caecum)	A barium meal and follow through → 1.narrowing of the lumen of 2.elevated caecum (up to subhepatic)
Treatment	1. In early cases → antituberculous drugs 2. In intestinal stricture → surgical excision	1. <u>Medical</u> by antituberculous chemotherapy. 2. <u>Surgical</u> by RT. hemicolectomy with removal of the diseased ileum in obstruction & if can't exclude malignancy



Intestinal ischaemia

Acute

Chronic

Acute intestinal Ischaemia

Definition

- Occlusion of one or more of the mesenteric vessels , usually the superior mesenteric artery or vein

- Acute intestinal Ischaemia is a very serious surgical emergency that is highly fatal.
- Commonly affect large intestinal segment but may be focal & affects a small segment.

Etiology

A- Acute mesenteric vascular occlusion:

1. Mesenteric arterial embolism (MAE).

- The source may be:

- AF with a left atrial clot.
- Mural thrombus after MI affecting the left ventricle.

The embolus usually lodges within few cm of the mouth of the SMA sparing the middle colic & few of the upper jejunal branches

2. Mesenteric arterial thrombosis.

- Due to mesenteric atherosclerosis (with history of intestinal angina)
- The extent of ischemia is more than that with embolism as the block is at the mouth of SMA sparing none of its branches.

3. Mesenteric venous thrombosis.

- Idiopathic or due to:

- Portal hypertension.
- Intra-abdominal sepsis.
- Hypercoagulable states.
- Intake of contraceptive pills.

B-Non-occlusive intestinal ischemia

- With low cardiac output states as arrhythmias & major sepsis.
- Splanchnic vasoconstriction → intestinal ischemia → gangrene.

Pathology

Damage occurs from the ischemia and re-perfusion

Ischemic damage

1. Within 3 hours of complete vascular block → mucosa sloughs, ulcerates & bleeds in the lumen.

Bacteria get access to the blood stream through the damaged mucosal barrier (bacterial translocation)

2. Within a few hours → the whole thickness of the intestinal wall is affected → exudes serosanguinous fluid in the peritoneum.

The patient may lose great amount of blood.

3. The intestine becomes gangrenous → perforates → peritonitis.

4. The gangrenous bowel loses peristalsis → acts as an obstruction

- The proximal intestine becomes distended with fluid and gas.

- When peritonitis develops → paralytic ileus → more distension.

Reperfusion damage

The return of blood flow (spontaneously or by surgery) → release of oxygen radicals → damages the cell membrane.

Clinical picture

Symptoms

- 1- Severe acute abdominal pain (main symptom)

Not respond to narcotics or nasogastric aspiration

- 2- Vomiting (with variable degrees)
- 3- bleeding per rectum.

Signs: -Early: little physical signs not matching the severity of pain.

- Later: blood loss → hypovolaemia and perforation → peritonitis so patient present with : a) general → shock

- b) Local → abdominal tenderness, rigidity, and distension

-Differential diagnosis:

1- Acute pancreatitis.

The common features are severe pain, vomiting, minimal local signs in the early phase and shock.

-The distinction is important to decide the management:

- a) Acute pancreatitis → conservative management
- b) Acute mesenteric vascular occlusion → urgent surgery.

2-Intestinal strangulation

- In non occlusive intestinal ischemia, there is no block to the mesenteric vessels, yet some patients have atherosclerotic narrowing

- The most sensitive layer to ischemia is the mucosa

-Type of patient:

1. Common in elderly due to thrombosis
2. May occur in young with source of embolism as AF

Investigations

Laboratory

- 1- CBC: a) Marked leucocytosis.
- b) Elevated haematocrit (except in massive blood loss)
- 2- Serum amylase:
Elevated in ½ the patients (not as high as in acute pancreatitis)

Radiological

- 1- Plain X-ray of the abdomen
- May show air-fluid levels in the proximal intestine.
- Lately, Intestinal necrosis show as intramural gas or gas in the portal venous system.
- 2- Arteriography
- Its value is controversial and may delay surgical intervention.
- If there are findings diagnostic of SMA embolism, preoperative infusion of papaverine directly in the artery improves intestinal viability.

- There is no specific or sensitive test to prove the diagnosis.
- The most useful aid is to put the condition in mind, particularly in the atherosclerotic or patient with cardiac arrhythmia.

Treatment

Urgent surgery is the key to survival.

Preoperative measures

1. Restoration of blood volume by blood and crystalloid infusion.
2. Parenteral antibiotics.
3. Nasogastric decompression by a Ryle's tube.

Laparotomy is done to:

1. Resect gangrenous intestine.

- a- Peritonitis with friable tissues
- b- Doubtful viability of the remaining intestine
- c- In patients with bad general condition

If present

- Stoma and an ileostomy bag.
- Bowel anastomosis is postponed until the general & local conditions improve

If absent

Primary anastomosis done & second-look operation after 24 hours is advised by some surgeons to check the viability

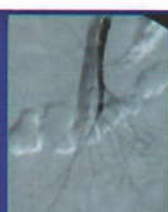
2. Restore arterial blood flow (in cases with reversible ischemia)

- a) In MAE → embolectomy
- b) In MAT → endarterectomy or bypass

3- In Mesenteric venous thrombosis

- Diagnosed at operation by → the presence of edema of the mesentery & extrusion of clots when mesenteric veins are cut.
- Treatment → resected gangrenous intestine

Pneumatosis Intestinalis (Gas in the wall of small bowel)



Postoperative management

The patient is given postoperative heparin & discharged home under oral anticoagulants for at least 3 months to prevent recurrence

Prognosis

- Mortality rate of:
 - a) Mesenteric venous thrombosis → 30%
 - b) Arterial occlusion → 45% (Embolism better than thrombosis)
- Resection of >70% of small intestine → short bowel syndrome

Chronic intestinal Ischaemia

Definition

Narrowing of the SMA and sometimes the coeliac artery

Etiology

1. Atherosclerosis (the **main** cause)
The intima of SMA is thickened at its mouth → narrowing.
2. Compression of the coeliac artery by the median arcuate ligament of the diaphragm (rare)

Clinical picture

Type of patient

Elderly patients who are likely to have atherosclerosis

Symptoms

- 1- **Abdominal angina** (the **main** symptom) (postprandial pain)
 - Starts 15-30 min after a meal & lasts for about an hour
- 2- The patient is usually afraid to eat → loses weight.

Signs

- 1- An upper abdominal bruit (in the majority of patients)
- 2- Clinical search to find atherosclerotic narrowing of other arteries.

Investigations

1. Aortography (**diagnostic**)

Particularly its lateral view which shows the narrowing of the origin of the artery & the presence of collateral circulation

2. CT angiography: is non-invasive and provides a 3D image.
3. Ultrasound and upper GI Endoscopy

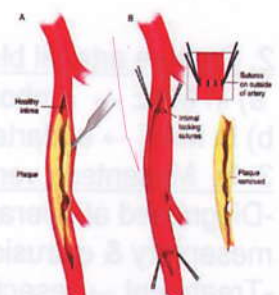
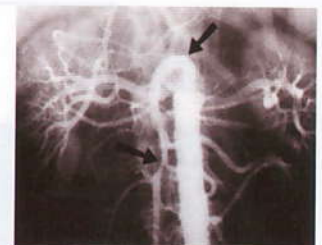
Exclude other causes of chronic abdominal pain

Treatment

- 1- Atherosclerosis is surgically treated either by endarterectomy (coring out the thickened intima) or by inserting a bypass dacron graft from the aorta to the SMA beyond the narrowing.
- 2- Median arcuate ligament compression → division of the ligament.

Short bowel syndrome

- Main feature: malabsorption.
- Many months are needed to accommodate by increasing the absorptive surface.
- Patients are fed:
 - a) Initially → TPN for a few months
 - b) Later → gradual oral intake is introduced, reducing the amount of parenteral feeding, until the patient can depend totally on normal oral feeding



Ischemic colitis

Etiology

- 1- A drop of blood pressure for any reason (the usual factor)
- 2- Surgery on the abdominal aorta if the IMA is sacrificed

Clinical picture

Type of patient

50-70 years old with chronic vascular disease of mesenteric vessels

Symptoms

Acute abdominal pain and the passage of blood per rectum

Site

The splenic flexure (the most commonly affected)

Investigations

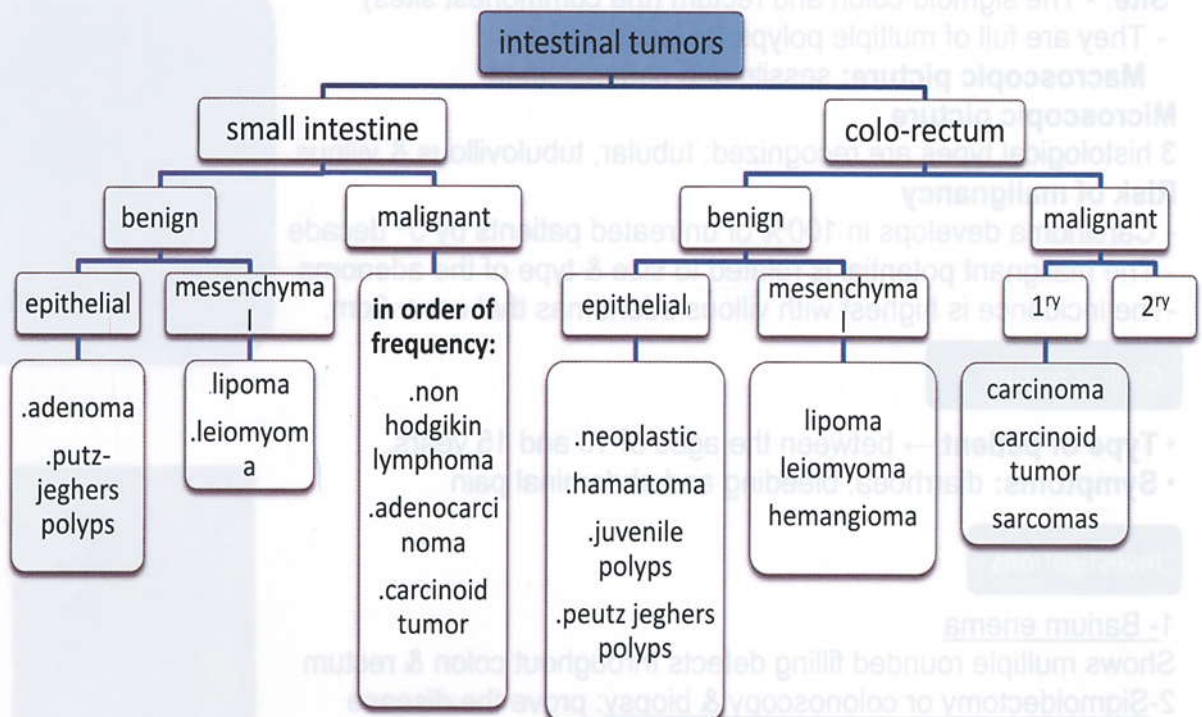
Plain X-ray → show **'thumb-prints'** due to edematous mucosa outlined by bowel gas.

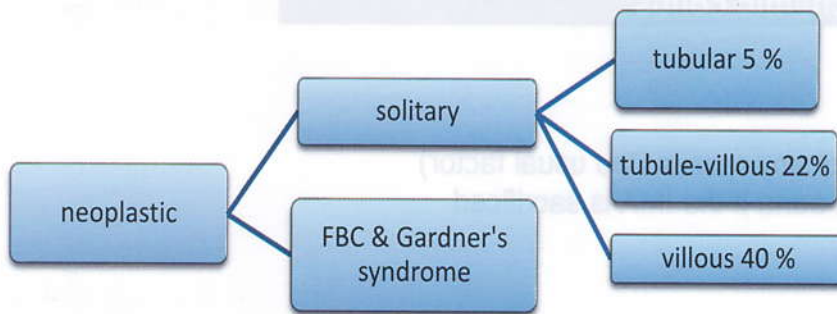
Consequences

- 1- Complete resolution.
- 2- Gangrene, perforation, and peritonitis.
- 3- Stricture

Treatment

- 1- Supportive by: IV fluids, analgesics & antibiotics.
- 2- Surgery (rare) for gangrene or stricture formation





- Small intestinal tumours are rare.
 - Benign tumours of SI may cause bleeding or intussusception
 - Carcinoid tumour of SI is less common than that of the appendix but more malignant
 - Tumours of the colon & rectum share Common properties so referred to as colo-rectal tumours.
 - Benign colorectal tumours usually form polyps.

Peutz-Jegher's syndrome

- Multiple familial intestinal hamartomatous polyps mainly affecting the jejunum.
- Melanosis of the oral mucous membrane and the lips.
- Not highly precancerous,
- Only the complicated polyps (causing bleeding or intussusception) require removal or excision of the affected intestinal segment

Familial polyposis coli (FPC)

Etiology

- An autosomal dominant disease.
- ($\approx 50\%$) of the children of affected parents has this disorder
- Only individuals with polyps transmit the disease.
- Males are affected more than females.

Pathology

- Site:** - The sigmoid colon and rectum (the commonest sites)
 - They are full of multiple polyps (at least 100)

Macroscopic picture: sessile and pedunculated

Microscopic picture

3 histological types are recognized: tubular, tubulovillous & villous.

Risk of malignancy

- Carcinoma develops in 100% of untreated patients by 5th decade
- The malignant potential is related to size & type of the adenoma.
- The incidence is highest with villous adenomas that are > 2cm.

Clinical picture

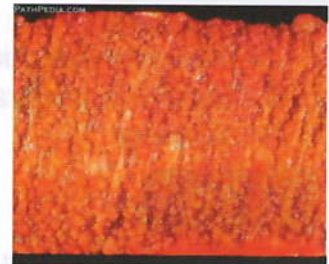
- **Type of patient** → between the ages of 10 and 15 years.
- **Symptoms:** diarrhoea, bleeding and abdominal pain

Investigations

1- Barium enema

Shows multiple rounded filling defects throughout colon & rectum

2- Sigmoidectomy or colonoscopy & biopsy: prove the disease nature



Gardner's syndrome is FPC with polyps in the rest of the GIT, osteomas of the mandible and skull, cysts, soft tissue tumours & desmoid tumours of the abdominal wall.



Treatment

- Is operative and is directed towards the eradication of the polyps.
- Surgical options are similar to those for ulcerative colitis, include:
 - 1- **Panproctocolectomy** (the **standard** treatment)
Excision of the whole colon and rectum with permanent ileostomy
 - 2- The rectum may be spared and an ileoproctostomy is performed with endoscopic fulguration of the remaining rectal polyps.
- Lifelong observation of the rectum is needed.
- 3- Proctocolectomy with distal rectal mucosectomy (the mucosa of the lower part of the rectum is cored out leaving its muscle wall)
 - An ileal pouch is fashioned to act as a reservoir & anastomosed to the anal canal within the remaining rectal muscles.

- The family members should be periodically examined by colonoscopy and similarly treated if they develop polyps.

- The avoidance of an ileostomy encourages the asymptomatic person to have the operation.

- Genetic screening of the relatives is performed nowadays.

Carcinoma of the colon and rectum

Incidence

- Is one of the leading causes of death in the Western society
- The peak incidence is in the 7th decade of life.

Etiology and risk factors

A-Chronic irritation

1. Ulcerative colitis (risk is highest with pancolitis >10 years)
2. Diet:
Lack of high fibre diet & ↑ animal fat (as in Western countries)
3. Uretero-colic anastomosis.

B-Benign lesions: Solitary villous adenoma.

C- Genetic factors

(Most colorectal cancers have an established genetic basis)

- Familial polyposis coli & Gardner's syndrome.
- The risk is **100%** in untreated cases.
- More in large adenomas **>2 cm** particularly of the villous variety.

The normal epithelium passes through several steps of genetic mutations to develop carcinoma (Multi-step theory):
Normal mucosa → dysplasia → adenoma → carcinoma

Pathology

Site

- 2/3 of cases in the rectum and the sigmoid colon.
- 10% occur in the caecum.
- The remainder is distributed over the rest of the colon.
- Multiple tumours occur in 5% of cases

Macroscopic picture

1. Cauliflower-like polypoid variety (more in the caecum)
2. Ulcerative type.
3. Stricture type (more common in the sigmoid colon)

Microscopic picture

- An adenocarcinoma that arises from the columnar epithelium.
- It may be well, moderately or poorly differentiated.
- A colloid structure (Some tumours that occur in younger patients & have a poor prognosis)



Spread

1- Direct spread

- Occurs 1st in the wall then to the neighbouring organs as UB
- The strong fascia of **Denonvillier** (lying in front of the rectum) delays the spread to the bladder.

2- Lymphatic spread

- produces deposits in the epicolic, paracolic, intermediate & then Superior or inferior mesenteric preaortic nodes
- Nodal involvement is found in **40%** of cases coming to operation.

3-Blood stream spread

- Produce liver metastases (in **20%** of patients coming to surgery)

4-Transperitoneal spread → peritoneal nodules & ascites



Staging

Tumor	Nodes	metastases
T1: Invades into submucosa	N0: No nodes involved	M0: No metastases
T2: Invades into musculosa	N1: 1-2 nodes involved	M1: Metastases
T3: Invades to the subserosa but not breaching the serosa	N2: 3 or more nodes involved	
T4: invades serosa or another organ		

Complications

1-Intestinal obstruction: in **20%** of cases particularly in left colon tumours. This is due to:

- The smaller lumen of the left colon
- Stool tends to be more solid.
- Carcinoma tends to be of the stenosing variety.

2- Perforation or the formation of an enterocolic or vesicocolic fistula.

3- Bleeding (the rule is chronic not massive bleeding)

4- Complications due to spread as: jaundice, liver failure & ascites.

Clinical picture

Type of patient

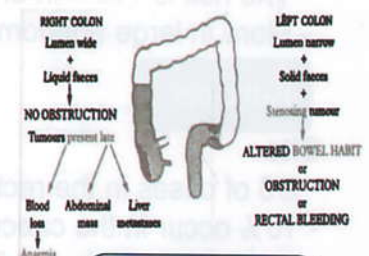
- Cancer rectum is commoner in males while cancer caecum is commoner in females.

-Clinical features depend upon:

The location of the tumour, its size & presence of metastases.

Right colon cancer

- Vague presentation with anaemia, weakness & loss of weight
 - Recurrent attacks of pain in the right iliac fossa.
 - A hard mass may be present in the right side of the abdomen.
- It is differentiated from appendicular mass by the long duration & absence of toxæmia and tenderness
- Does not present by I.O. as:
 - The lesion is usually of the cauliflower variety
 - The contents are liquid
 - the lumen of the colon is wide.



3A → Anaemia,
Anorexia,
Asthenia

I.O. occurs in right
colon cancer only if
lesion obstructs
ileocaecal valve

Left colon cancer

1. Change of bowel habits (the usual presentation)
 - Usually as progressive constipation but may be diarrhoea or attacks of constipation alternating with diarrhoea.
 - are usually diagnosed & treated as having colitis.
 - Spurious diarrhoea (early morning slime) may be present.
2. Large bowel obstruction.
 - may present as acute, subacute or chronic obstruction.
 - There is constipation, severe distension but vomiting is late.
 - Sigmoid colon cancer is a very common cause of I.O. in elderly
3. Bleeding per rectum.
 - is a common cause of fresh bleeding per rectum but not a massive
4. Mass in the left side of abdomen.
 - Rare as the lesion is usually infiltrating schirrous
 - When stools get impacted above the obstruction → palpable mass

Rectal cancer

1. Bleeding per rectum (usually slight)
2. Tenesmus (sense of painful incomplete evacuation) & the passage of mucus.
3. Painless (usually) unless it has spread outside the rectal wall or infiltrated the anal canal.
4. DRE → palpation of lesions within 10 cm of the anal verge.
 - Higher tumours are revealed by sigmoidoscopy

Investigations

For diagnosis

1. Sigmoidoscopy
 - Is essential in all patients with altered bowel habit or rectal bleeding
 - Mandatory in patients >40 who complain of piles.
 - Biopsy is obtained from suspicious lesions
 - Biopsy from a higher tumour can be obtained by the colonoscopy

2. Barium enema.

- The tumour appears as a fixed irregular filling defect.
 - Annular strictures of LT colon show → **"apple core appearance"**
 - exclude a 2nd higher tumour even if rectal cancer is palpable
3. For patients with acute I.O., to assure the diagnosis →
 - a) Plain X-ray of the abdomen
 - b) Blood picture, blood urea & electrolytes are needed.
 - c) Barium enema

For staging

1. Transrectal ultrasonography or rectal protocol magnetic resonance imaging (MRI)

- Evaluate depth of invasion, the circumferential resection margin (CRM) & lymph node status
 - help determine the need for preoperative chemoradiation therapy
2. Abdominal ultrasound or CT scan
 3. Chest X-ray
 4. IV urogram → If tumour is expected to be close to ureter

- Massive bleeding per rectum occur in: diverticular disease & angiomatous malformation

- Whenever rectal bleeding occurs in a middle aged or older individual (even in the presence of haemorrhoids) coexisting rectal cancer must be ruled out.



Preoperative: 1- Liver function tests

2-CBC → may reveal microcytic hypochromic anaemia

For follow up

• Carcinoembryonic antigen (CEA)

- Is a non specific tumour marker → high level in colorectal cancer
 - It is of prognostic rather than diagnostic value.
- The level drops after a successful radical surgery.
- If it shows a rise in the follow up period = recurrence.

Treatment

General principles

- Surgery is the main line of treatment.
- Radical resection is the only curative measure.
- For inoperable tumours resection also offers the best palliation.
- Treatment depends on whether the tumour presents by acute obstruction or not and whether the tumour is operable or not

I -No acute obstruction

A-Operable (potentially curable) cases

1- Preoperative colon preparation allows a safer anastomosis

2- Should receive elective radical resection (aim is to cure)

- This entails:

- a) Removal of the tumour bearing segment
- b) Removal of Lymphatic drainage area
- c) Restore bowel continuity.

In one mass

Site	Operation	Divided vessels
caecum	RT hemicolectomy	Ileocolic & right colic vessels
ascending colon or hepatic flexure	extended RT hemicolectomy	1. Ileocolic 2. RT. colic 3. middle colic
transverse colon	Transverse Colectomy	middle colic vessels
left colon	left hemicolectomy	inferior mesenteric
sigmoid colon	Sigmoid colectomy	sigmoid vessels (at origin from IMV)
upper 1/3 of rectum	anterior restorative resection	inferior mesenteric artery (LT colic may be spared)
Middle & Lower 1/3 of the Rectum	Neoadjuvant chemoradiation followed by anterior restorative resection	inferior mesenteric artery (LT colic may be spared)

- Inoperability can be judged before the operation by the presence of liver Metastases on US examination

- Still tumours that are preoperatively judged to be operable may prove inoperable during exploration, because of liver metastasis, peritoneal nodules or fixation of the tumour to important irremovable structures (irresectable tumour)
- Colectomy may be done open, laparoscopically or robotically

(Laparoscopic approach is proven oncologically equal to open surgery with the benefit of shorter recovery "Lancet Oncol")

- At least 12 nodes are removed to ensure appropriate staging.
- Must ensure:
 - a) Adequate proximal & distal margin
 - b) High ligation of the arterial pedicle for LN clearance
 - c) Tension-free anastomosis & good blood supply to the anastomosis or stoma.

Neoadjuvant chemoradiation consisting of:
 5-FU, leucovorin with concomitant radiation therapy (XRT, 54 cGy)
"Currently standard for all patients with T3 or T4 lesions or node positive disease on imaging (TRUS or MRI) "

• The goal of rectal surgery:

- 1- Remove the cancer with adequate margins
- 2- Total mesorectal excision (TME)
- 3- LN clearance with high ligation of the arterial pedicle (IMA)
- 4- Consideration of future continence & urogenital function.

• Tumours of the middle and Lower 1/3 of the rectum used to be treated by abdominoperineal resection and a colostomy, the change towards anterior restorative resection is due to:

- 1- Realization that a 2 cm safety margin in the rectum below the tumour is compatible with radicality, thus leaving enough distal rectum for anastomosis.
- 2- The development of the circular staplers markedly facilitated this technically difficult procedure.

• Abdominoperineal Excision with Terminal Left Iliac Colostomy still done if there is:

- 1-sphincter involvement
- 2- Incontinence
- 3- Improper distal Safety Margin (< 2 cm)
- 4- Aggressive pathology (Undifferentiated, Mucinous or Colloid types of cancer lower rectum).

-The following are removed in RT hemicolectomy:

1. Terminal 10 inches of ileum
2. Caecum
3. Ascending colon
4. Hepatic flexure
5. RT half of the transverse colon
6. The peritoneum of the posterior abdominal wall between the colon and the superior mesenteric (contains the divided arteries, veins & lymph vessels and nodes).

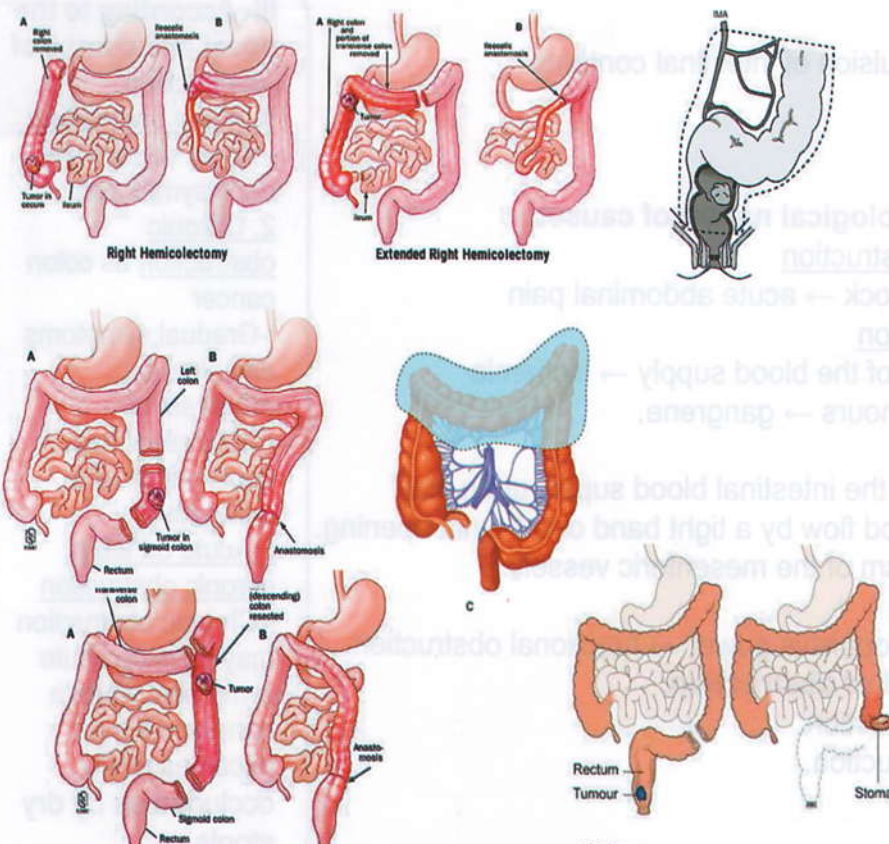
(Operation is followed by doing ileotransverse anastomosis)

- In extended RT hemicolectomy → resection extends to the junction of RT 2/3 & LT 1/3 of transverse colon.

- In transverse Colectomy → transverse colon is resected with both the hepatic & splenic flexures, the transverse mesocolon & the omentum

- LT hemicolectomy → the resection extends down to the rectum

- in anterior restorative surgery → the sigmoid colon & upper 1/2 of rectum excised



B-Inoperable (incurable) cases

(Whether inoperability is diagnosed before or during operation)

1-**Respectable tumor** → palliative local resection of the colon cancer without lymphatics and vessels removal

- Aim: is to avoid the risk of obstruction & bleeding.

2- **Irresectable tumor** → operation to avoid obstruction.

a) Right colon → a side to side ileotransverse anastomosis.

b) Elsewhere in the colon → a proximal colostomy.

- The rectum show partial response to local radiotherapy.

- Supplementary chemotherapy with 5-fluorouracil (5-FU) may be useful for colon cancer.

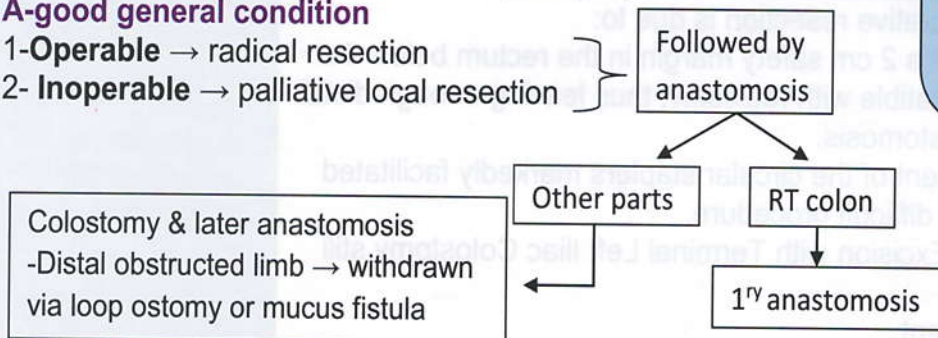
II- Acute intestinal obstruction

-Urgent surgery is required after adequate preparation

A-good general condition

1-**Operable** → radical resection

2- **Inoperable** → palliative local resection



Worse prognosis of colorectal cancer occur in:

1. Young age at clinical presentation.
2. Colonic obstruction or perforation.
3. Blood vessel or lymphatic invasion.
4. Liver &/ or distant metastases.
5. Aneuploidy

B- Critically ill → proximal colostomy

Intestinal obstruction

Definition

Arrest of downward propulsion of intestinal contents

Classification

I-According to the pathological nature of cause:

1. Simple mechanical obstruction

Caused by an organic block → acute abdominal pain

2. Strangulation obstruction

- Significant impairment of the blood supply → ischemia

- If not relieved within 6 hours → gangrene.

- may result from:

a) **Volvulus** = twisting of the intestinal blood supply upon itself

b) Constriction of the blood flow by a tight band or a hernia opening.

c) Thrombosis or embolism of the mesenteric vessels.

3. Paralytic ileus

Is due to loss of bowel propulsive power → functional obstruction

II-According to the level of obstruction:

1. High small bowel obstruction.

2. Low small bowel obstruction.

III- According to the onset and course of obstruction:

1. Acute obstruction.

- Rapid course and early symptoms

2. Chronic obstruction as colon cancer

- Gradual symptoms and slowly progressive.

- The patient has constipation and distension.

3. Acute on top of chronic obstruction

- Chronic obstruction may develop acute symptoms when a narrowed lumen becomes totally occluded as by dry stools

Acute mechanical intestinal obstruction (general principles)

Causes

1. In the lumen:

Faecal impaction, gallstone and parasitic infestation

2. In the wall

Congenital atresia, tumours, Crohn's disease, chronic diverticulitis and mesenteric vascular occlusion

3. Outside the wall

Adhesions (commonly postoperative), strangulated hernia & volvulus

Pathology

I-Simple obstruction

a) Distal to the obstruction

The intestine empties and becomes collapsed.

b) Proximally

1-the intestine becomes distended by :

(i) Gas due to:

- Swallowed air (68%)
- Diffusion from congested vessels (22%)
- Bacterial fermentation (10%).

(ii) Fluid due to: GIT secretions (8 liters / day)

- As the intraluminal pressure rises → absorption ceases

2- Hyperperistalsis

- Due to stretch of smooth muscles to overcome the obstruction
- Increased both power and frequency of contractions.

3-Impaired blood supply

- Due to distension
- Lead to ulceration and perforation.
- Examples :
 - (i) Closed loop obstruction (colon obstruction with a competent ileocaecal valve), when pressure rises in the closed segment → perforation of the caecum
 - (ii) Pressure of an adhesion band or the edge of a hernia defect on the bowel wall → local ischemic necrosis → Perforation

II- Strangulation obstruction , In addition to the above:

- Unrelieved strangulation → ischemia → gangrene → Perforation → bacteria & toxins reach peritoneal cavity → peritonitis
- Unrelieved strangulation can lead to septicaemic shock
- The mucosa is the 1st layer to suffer from ischemia → acute ulceration & intra-luminal bleeding ,If a long bowel segment is involved → massive blood loss

Common causes according to age

1. Neonates:

Congenital atresia, volvulus neonatorum, anorectal malformations, meconium ileus & Hirschsprung's disease

2. Infants:

ileocaecal intussusception, Hirschsprung's Disease & strangulated hernia

3. Adults:

Adhesions & strangulated hernia.

4. Elderly: Colon carcinoma & strangulated hernia.

• Strangulated hernia is a common cause in different age groups

- The most common cause of S.I. obstruction in:

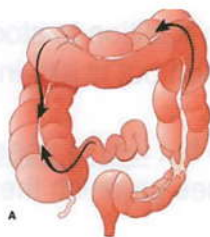
a) Adults

→ adhesions

b) Children

→ strangulated hernia

- The most common cause of large bowel obstruction is colon cancer



Lethal effects

- 1-Fluid and electrolyte loss
(From vomiting & accumulation in the proximal bowel)
- 2- Septicaemia (from peritonitis)

Clinical picture

Symptoms

1. Pain (the 1st symptom)

- It is colicky & caused by the hyperperistalsis

2. Distension

- a) In colon obstruction → marked & present mainly in the flanks.
- b) In high obstruction → less marked or absent.
- c) In low small bowel obstruction → central abdominal distension

3. Absolute constipation

- It is failure to pass flatus in addition to stools
- It is early in colon obstruction but is late in high obstruction.

4. Vomiting

- is early in high obstruction but late or absent in colon obstruction
- In neglected cases becomes greenish then brown & offensive i.e faeculent (not faecal).

Signs

I-General examination.

Evidence of dehydration as: tachycardia, oliguria, dry tongue or even hypotension.

II- Abdominal

a) Inspection

- 1- Distension & visible peristalsis.
- 2- Look for strangulated external hernia
- 3- Scars of previous abdominal surgery
(May denote intra-abdominal adhesions)

b) Palpation → A mass may be felt (tumour or intussusception).

c) Auscultation → Accentuated intestinal sounds.

d) DRE

- Reveals an empty rectum.
- An exception is finding a hard faecal mass in an elderly bed-ridden patient causing faecal impaction.

Questions to answer for clinical assessment of I.O

1. Is there intestinal obstruction?

I.O. is diagnosed by the presence of colicky abdominal pains, distension, Vomiting and absolute constipation.

2. What is the pathological type of the obstruction?

Strangulation is suspected when there is : severe pain (not relieved by bowel decompression) , toxæmia, abdominal tenderness & rigidity

In paralytic ileus → no pain & the bowel sounds are absent.

3. What is the level of obstruction?

- a) In high small bowel obstruction → early vomiting & dehydration
- There is slight abdominal distension.

- Pain, distension, vomiting & absolute constipation are the cardinal symptoms of acute mechanical I.O. but not necessarily present in all cases

-Strangulation needs urgent surgery, it is suspected in:

Symptoms:

Pain that is not relieved by NG suction
(Pain is partly caused by hyperperistalsis & partly by ischemia, decompressing the bowel does not relieve ischemic pain)

Signs:

General → Toxic patient, fever, leucocytosis & evidence of blood loss (pallor, tachycardia, & hypotension)
Local → Marked tenderness, rebound tenderness & rigidity

Investigations are needed to confirm the diagnosis Of I.O., to define its level & cause and to estimate the severity of water & electrolyte imbalance

b) In low small bowel obstruction → delayed vomiting (for 12 hours) and there is central abdominal distension.

c) In colonic obstruction → early constipation while vomiting may be absent or occurs after few days.

-Distension is marked especially in the flanks.

4. What is the cause of obstruction?

Suspected from the age of the patient & by the presence of certain physical signs as presence of an appendectomy scar → adhesive intestinal obstruction

Investigations

Radiological

1. Plain X-ray of the abdomen.

Erect film → shows multiple gas-fluid levels (confirm the diagnosis)

Supine film → shows the level by the gas pattern of the distended proximal intestine :

- Distended jejunal loops → characteristic circular mucosal folds (**valvulae conniventes**) crossing from one side of the lumen to the other.
- Distended ileal loops → featureless tubes with no mucosal pattern
- A colon full of gas → haustrations that do not reach the other side of the lumen

2. Ultrasound examination

-Can reveal distended bowel loops.

-A mass of intussusception can also be demonstrated.

3. CT scan with contrast

- Has a sensitivity of **80-90%** for the diagnosis of I.O.

Laboratory

- CBC
- Blood urea and electrolytes.

Treatment

Urgent relief of obstruction by surgery after adequate preoperative preparation

I-Pre-operative preparation "Drip and Suck"

1- IV line:

- Replacement of fluid and electrolytes
- Blood & plasma if the patient is shocked.
- Ringer's lactate solution is needed.
- Antibiotics are given if there is a possibility of strangulation

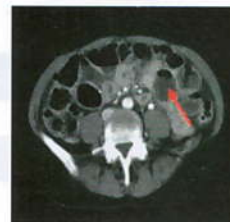
2- Gastric aspiration by NG tube:

- To decompress the bowel
- Reduce the risk of inhalation during induction of anaesthesia.

3- A Foley catheter → to check the urine output

II- Operation

1- A longitudinal exploratory incision is performed except in strangulated hernia → the incision is directly over it.



Non-viability is known by:

1. Loss of peristalsis
2. Loss of normal lustre.
3. Colour change → greenish or black bowel is non-viable (purple bowel may still recover)
4. Loss of pulsations in the mesentery

2- Exploration (1st is to look at the caecum)

- a) If collapsed → small gut obstruction.
- b) If distended → large bowel obstruction.

3- Determine the level of obstruction which

Is the junction of dilated and collapsed bowel loops

4-Relief of obstruction

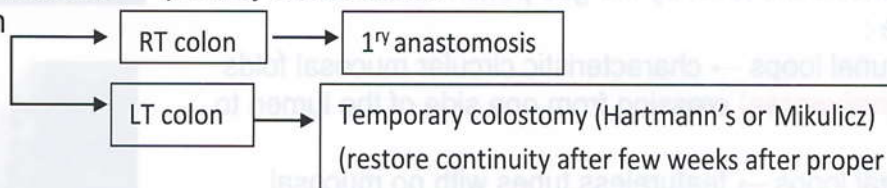
By division of adhesive bands, reduction and repair of a hernia or untwisting of volvulus

5- Assess bowel viability

- a) Viable → reduced to the abdomen
- b) Doubtful bowel → wrap it in hot packs for a few minutes (may recover)
- c) Non-viable intestine → resection then according to site :

i- Small intestine → primary anastomosis

ii- Colon



VIALBLE BOWEL



NON VIALBLE BOWEL

III- Conservative treatment

- 1. Adhesive intestinal obstruction → IV drip & NG suction.
- 2. Ileocaecal intussusception → a barium enema(has hydrostatic effect)& the reduction is radiologically monitored on the screen.
- 3. Sigmoid volvulus → a rectal tube passed through a Sigmoidoscope can untwist the obstructed segment.
- 4. Faecal impaction → enema (dissolve the obstructing hard faecal mass)

-Indications of conservative treatment

- 1. Case is early
- 2. There is no clinical evidence of intestinal ischemia.
- Failure of conservative ttt & suspicion of strangulation → surgery indicated

Acute mechanical intestinal obstruction - Special forms

Neonatal intestinal obstruction

Discussed in pediatric surgery

Intussusception

Definition

invagination of an intestinal segment (**intussusceptum**) into the lumen of an adjacent one (**intussusceptient**).

Types

1. Ileo-ileal.

A loop of ileum invaginates itself into an adjacent ileal loop.

2. Ileo-caecal. (the commonest type & usually affects infants)

The terminal ileum invaginates into the colon with the ileo-caecal valve repressing the apex of the intussusception

An intussusception is composed of

- 1. An inner tube & a returning tube → Intussusceptum
- 2. An outer tube → Intussusceptient
- The blood supply of the inner layers of the intussusception is liable to be impaired at the neck of the intussusception.

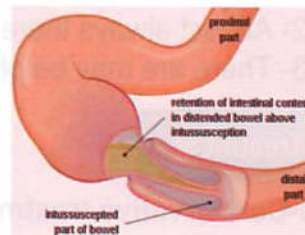
3. Ileocolic.

An ileal loop invaginates into an ileal loop and then passes to the colon through the ileocaecal valve.

The blood supply of the ileal loop is tightly compressed by the ileocaecal valve.

4. Colo-colic

A loop of colon invaginates into an adjacent colonic segment

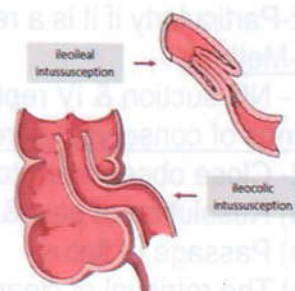


Colo-colic



Ileo-caecal

Etiology



In adults there is an evident cause at the head of the intussusceptum as:

1. Polyp
2. Meckel's diverticulum
3. Submucous haematoma in Henoch-Schonlein purpura.

Adhesive intestinal obstruction

Incidence

The commonest cause of intestinal obstruction in adults in developed countries

Etiology

1- Postoperative (the commonest)

-Exact etiology is obscure.

-It is suggested that adhesions develop at the sites of ischemia of the bowel or at the obstruction scars of the abdominal wall.

2- Post-inflammatory

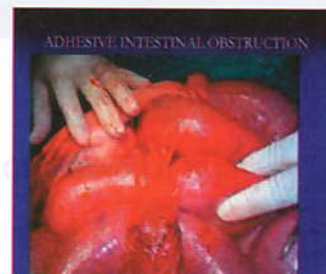
Follow previous septic or tuberculous peritonitis.

Pathology

- 1- Adhesions may be multiple or solitary.
- 2- They bind one intestinal loop to another or to abdominal wall.
- 3- They induce obstruction by :
 - a) Kinking
 - b) directly obstructing a small intestinal loop.
- 4- Strangulation may result from :
 - a) Compression of the loop's blood supply.
 - b) band → direct pressure on the intestine at the level of block → localized ischemic necrosis
- 5- Adhesions have a tendency for recurrence producing recurrent intestinal obstruction.

Clinical picture

1- Acute or recurrent acute small bowel obstruction → (colicky abdominal pain, vomiting, distension & absolute constipation)



- 2- Almost always there is a scar of previous abdominal surgery.
- 3- There may be physical findings of strangulation.

Treatment

I-Conservative treatment

-Indications:

- 1- In early cases with no evidence of strangulation
- 2- Particularly if it is a recurrent obstruction

-Method:

- 1- NG suction & IV replacement of losses (drip and suck) → main lines of conservative treatment.
- 2- Close observation to judge success which is indicated by:
 - a) Resolution of pain & distension
 - b) Passage of flatus
 - c) The retrieval of clear gastric aspirate.

II-Surgery

-Indications:

- a) The above trial fails
 - b) There is evidence of strangulation or gangrene.
- At operation → Adhesions are divided and the bowel viability is assessed and dealt with accordingly

Conservative treatment should not be prolonged if there is no response in 48 hours

Volvulus

Definition

Twisting of a bowel loop around its mesenteric axis

Pathology

- 1- Produces a combination of:
 - a) Closed-loop obstruction
 - b) Occlusion of the main vessels at the base of the involved mesentery (**strangulation**)
- 2- In a closed loop, the pressure rise rapidly → gangrene & perforation.
- 3- Commonest site is the sigmoid colon (may affect the caecum, stomach or S.I.)

Clinical picture

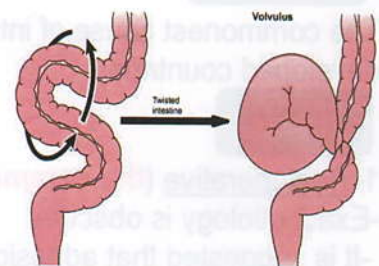
Type of patient

Commoner in elderly chronically constipated males.

Symptoms

- 1-Colicky pain
- 2- Marked distension (due to accumulation of fluid & gas in the closed sigmoid loop)
- 3-Absolute constipation
- 4- Vomiting

Neglected cases show evidence of peritonitis



-Predisposing factors:

1. A long sigmoid colon.
2. A narrow base of sigmoid mesocolon.
3. A heavily loaded sigmoid as a result of chronic constipation.
4. Adhesions at the apex of the sigmoid loop → facilitate its twisting

Signs

- 1-Inspection → marked distension
- 2-Palpation → tense balloon of volvulus
- 3-Percussion → tympanic resonance
- 4- Auscultation → mad abdomen
- 5- DRE → empty rectum

Investigations

1. Plain X-ray

Omega loop → the huge gas-filled sigmoid loop that may look like the inner tube of a car tyre

(The base of the distended loop points to the LT lower abdomen)

2. Laboratory: CBC, Urea & electrolytes



Treatment

I-Conservative treatment

-Indications: in early cases with no evidence of gangrene.

-Method:

-Rectal tube is passed through a sigmoidoscope → untwist the sigmoid loop.

-Success is known by → a gush of gas & fluid stools.

-The tube is left in place and the patient is prepared for later elective resection of the long sigmoid to prevent recurrence.

II- Surgery

-Indications:

- a) Failure of the conservative treatment
- b) Late presentation.

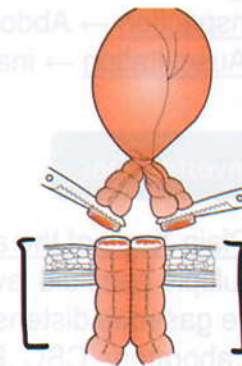
-Method:

1-If colon is gangrenous → resection

(The proximal colon end is brought out to the skin as a terminal colostomy & the distal end is closed by sutures "Hartmann's Procedure" for later elective anastomosis)

2- Viable sigmoid → is untwisted and if:

- (i) Short → fixed to the posterior abdominal wall
- (ii) Long → resected (as for gangrenous cases)



Paralytic ileus

Definition

It is failure of the neuromuscular mechanism → failure of the peristaltic waves of the intestine (adynamic obstruction)

Etiology

1-Reflex inhibition of intestinal motility following:

- a) Major abdominal operations (**most common**)
- b) Spine fractures
- c) Hyperextension of the spine (in plaster jacket)
- d) Retroperitoneal haemorrhage.

-Some degree of bowel atony follows abdominal operations for 24-48 hours.

-Paralysis that extends > 3 days deserves search for another cause (hypokalaemia or leaking anastomosis → peritonitis).

-The duration of postoperative reflex ileus depends on bowel manipulation & irritation by blood, bile, urine or pus.

-The exact mechanism not known but probably due to **sympathetic overaction**.

2. Metabolic abnormalities

As hypokalaemia, uremia and diabetic ketoacidosis

3. Peritonitis

-Induces direct toxic effect on the nerve plexuses of the intestine.

-May be complicated by the formation of fibrinous adhesions → kink the intestinal loops (adding mechanical obstruction)

4. Drugs as:

a) Anticholinergics (e.g., propanthine)

b) Tricyclic antidepressants (if taken in large doses)

Clinical picture

Symptoms:

1-Abdominal distension

2- Absolute constipation

3- Effortless vomiting

Signs:

1-Inspection → Abdominal distension

2- Auscultation → inaudible intestinal sounds (**silent abdomen**)

Investigations

1. Plain X-ray of the abdomen

- Multiple gas fluid levels.

-The gaseous distension includes the whole small & large intestines.

2. Laboratory: CBC ,Blood urea & electrolytes

Prevention

1. Preoperative

- Prevention & correction of biochemical disturbances.

- Hypokalaemia is prevented & treated by IV potassium (guided by serum level)

2. Intraoperative → Gentle handling of the intestine

3. Postoperative:

-For major abdominal surgery, a NG tube to decompress the bowel

Treatment

1. IV replacement of the lost fluids and electrolytes & NG suction (**Drip and suck**) "Essentials"

2. Correction of metabolic abnormalities & hypoproteinaemia.

3. If a postoperative ileus is prolonged (>3 days) → think of:

a) Peritonitis from a leaking intestinal anastomosis

b) Mechanical obstruction from early fibrinous adhesions.

4. The above measures are usually successful but occasionally in resistant cases, use → a parasympathomimetic as prostigmine

-Pathology:

1- Marked distension of the small & large intestines with gas & fluid.

2-The patient suffers a severe loss of fluid and electrolytes in:

a) The distended bowel

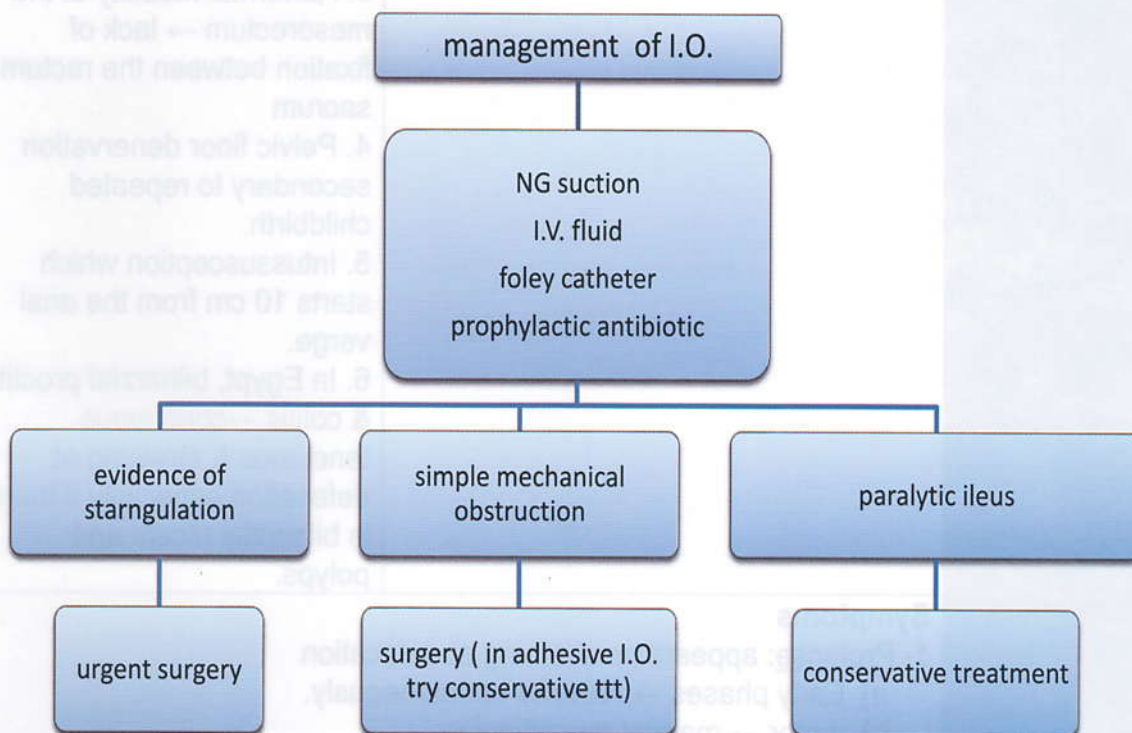
b) Through vomiting

-The patient has no colicky abdominal pains but there is only a sense of fullness and discomfort.

- There may be evidence of localized or generalized peritonitis



Necessitate reoperation.



Rectal prolapse

Definition

The protrusion of the rectum through the anus

	Partial (mucosal) RP	Complete RP
Definition	mucosa only prolapses	whole thickness of the rectal wall is prolapsed
Incidence	common in children	more in the elderly (F > M) In Egypt, males affected more due to bilharziasis of rectum
Etiology	A- In children 1. Loss of the curve of sacrum, the anus & rectum → a vertical tube. 2. Loss of weight results In loss of the supporting pararectal & ischiorectal fat. 3. Straining at defecation due to prolonged diarrhea or whooping cough B-in adults due to: 1. Advanced cases of haemorrhoids. 2. urethral stricture → continuous straining 3. Atony of the sphincter in the elderly. 4. Iatrogenic injury to the anal musculature during a fistula operation.	The exact etiology unknown. Many theories are suggested: 1. Failure of rectal support by the levators & pelvic fascia due to defective collagen maturation in the pelvic floor. (Excessive stretching of these structures during labour may explain its association with childbirth) 2. may represent a sliding type of hernia (most patients have a deep rectovesical or rectovaginal extensions of the Douglas pouch)

	3. Abnormal mobility of the mesorectum → lack of fixation between the rectum & sacrum 4. Pelvic floor denervation secondary to repeated childbirth. 5. Intussusception which starts 10 cm from the anal verge. 6. In Egypt, bilharzial proctitis & colitis → continuous tenesmus & straining at defecation especially if there is bilharzial ulcers and polyps.	
Clinical features	Symptoms 1- <u>Prolapse</u>: appears on straining at defecation a) Early phases → reduces spontaneously. b) Later → manual repositioning. 2- Mucus discharge, bleeding from the congested mucosa & discomfort 3- A degree of faecal incontinence (common with complete RP) Is the distressing symptom that urges the patient to seek medical advice 4- complications as :Irreducibility, ulceration & gangrene Signs 1- patient is asked to strain to demonstrate his/her complaint (squatting position is required if the prolapse does not show in the lateral position) 2- The anal sphincter tone is also assessed (palpation)	
Pathology		
a) length	< 5 cm	> 5 cm
b) mucosal corrugation	Absent	Often present
c) thickness	Mucosa	Whole rectal thickness
Differential diagnosis	1. Prolapsing haemorrhoids. 2. Prolapsing polyp. 3. Prolapsing intussusception.	
Treatment	A-Children -Surgery is rarely indicated (condition resolves spontaneously with age) <u>-Instructions :</u> 1- Avoidance of constipation & straining. 2- Improving the nutritional status 3- Manual reduction after defecation followed by strapping of the buttocks to prevent prolapse (postpone child training to defecate in the potty) 4- Submucous injection of 5% phenol in almond oil (in resistant cases)	

B- Adults

I-Mucosal prolapse.

- 1- early cases → submucous injection of 5% phenol in almond oil
- 2- in large prolapse → excision (similar to haemorrhoidectomy)

II-Complete prolapse → requires special surgeries as:

1. Rectopexy.

- Through abdominal approach → the rectum is mobilized & pulled up
- A rectangular piece of polypropylene mesh or ivalon sponge is attached by sutures to the presacral fascia then wrapped & sutured around the back and sides of the rectum.
- The mesh initiates fibrous tissue → fixes the rectum in its new position.

2. Ventral rectopexy (a newer option)

- The anterior plane is mobilized, a permanent mesh is secured to the anterior rectal wall at the level of pelvic floor, then the mesh is anchored to the sacral promontory.
- Advantages : lower complication rates, similar recurrence rates & improved functional outcomes

3. Sigmoid resection and rectopexy (FrykmanNGoldberg procedure)

- shortens the redundant recto-sigmoid colon with posterior sacral fixation.

- Recurrence <10% following rectopexy with or without resection.

4. Perineal proctectomy (modified Altemeier procedure)

- Alternative for patients with severe anal incontinence due to complete eversion and stretch of the anal canal.
- Recurrence rate is generally around 20%

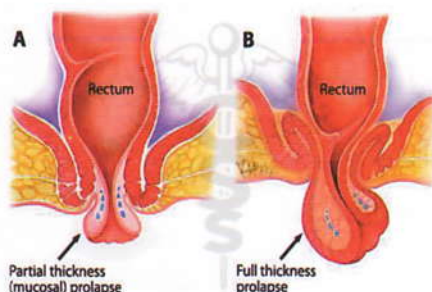
5. Delorme's operation

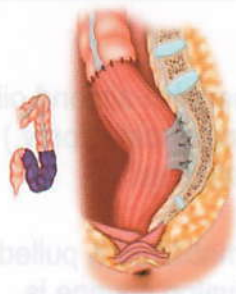
- Done through a transanal approach.
- Circumferential excision of excess rectal mucosa above the dentate line
- Sutures pass through the rectal muscle, plicating it to approximate the upper edge of the remaining mucosa to that at the dentate line

6. Narrowing of the anus (Thiersch's operation)

- Indication : frail patient who are unable to stand a major surgery.
- The prolapse is reduced & a stainless steel wire or a thick monofilament suture is tunneled subcutaneously around the anus.
- The suture is then tied to fit around the finger of the surgeon.
- the simplest operation but provides the poorest results.

(The results of operations are clouded by a proportion who get a recurrence and by those who continue to have faecal incontinence)

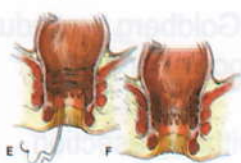
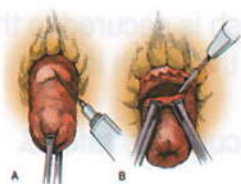
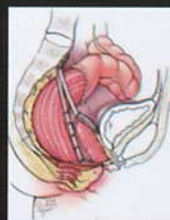




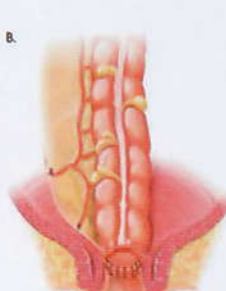
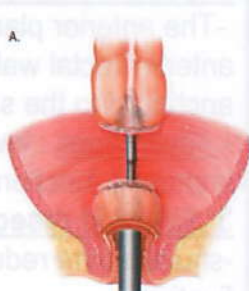
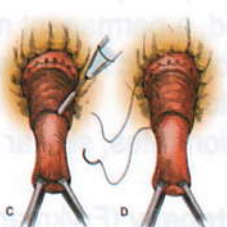
Sigmoid resection & Rectopexy



Anterior ventral rectopexy

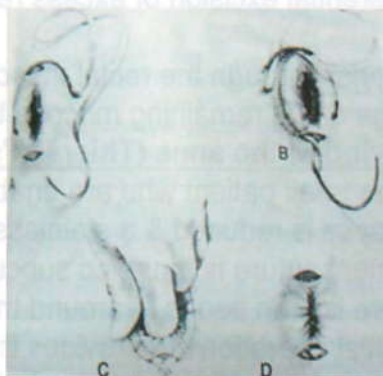


Delorme's operation



Perineal proctectomy

Thiersch's operation



The anal canal

Anal Physiology

Physiology of continence

The factors that maintain continence to stools and flatus are:

1. Anal sphincters

a) The internal anal sphincter (the **major** determinant)

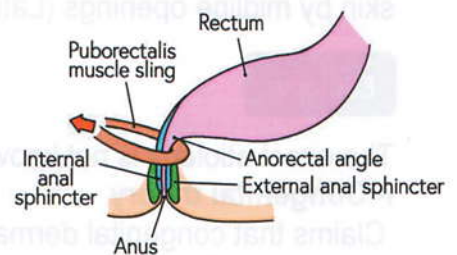
- Contribute **80%** of the resting pressure within the anal canal.
- Is under autonomic control
- Inhibited by → the parasympathetic
- Stimulated by → the sympathetic nervous system.
- It principally controls the continence to flatus.

b) The external sphincter

- Contribute **20%** of the resting pressure within anal canal
- Is a striated muscle supplied by somatic nerves.
- Its Division during fistula surgery → minor functional disability.

2. Puborectalis and anorectal angle

- The puborectalis muscle forms a sling that pulls the anorectal junction forwards to maintain its **right angle**
- Electromyography revealed a continuous basal resting activity that is controlled by spinal reflexes with no conscious effort (even during sleep).
- Loss of spinal reflexes in spinal cord injury or damage of the puborectalis → faecal incontinence.
- If intra-abdominal pressure is raised (by coughing or lifting heavy objects) → anterior rectal wall compresses onto the upper anal canal → occludes its lumen.



3. Sensation in the rectum

- Produce awareness of filling which contributes to continence.
- The stretch receptors for this sensation are located in the levator ani muscle and external anal sphincter.

- The mucosal lining of the upper part of anal canal contains receptors that discriminate between flatus & faeces.

4. Neuromuscular reflexes in the pelvic floor & external sphincter.

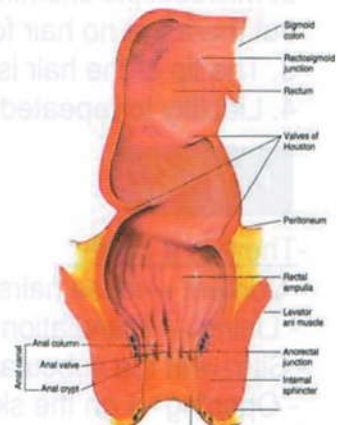
- There is a spinal reflex between the rectus muscle on one hand, and the pelvic floor & external sphincter on the other.
- contraction of the abdominal wall muscles during any form of straining → intra-abdominal pressure rises → anal sphincters & pelvic floor muscles contract → prevent stress faecal incontinence.

5. Folds of Houston

- These are folds of rectal mucosal membrane
- Manometry suggests that they have a minor role in continence.

6. Anal cushions

- Formed by the terminal branches of the superior haemorrhoidal vessels



Mechanism of defecation

1. Rectal distension (by stools or flatus) → the internal sphincter relaxes → a small part of rectal contents enter the upper anal canal.

2. Sampling reflex

The sensitive epithelium in upper anal canal can differentiate its type whether being flatus, liquid or solid stools

3. According to circumstances:

A) If convenient → the subject relaxes the external sphincter & the puborectalis → widening the anorectal angle

- Straining will expel the rectal contents to the outside.

B) If not convenient → the puborectalis and the external sphincter contract → push the sample of luminal contents back up.

Pilonidal Sinus

Definition

A subcutaneous granulating cavity originally described as a nest (Latin-**nidus**) containing hair (Latin-**pilus**) and connected to the skin by midline openings (Latin-**sinus**)

Etiology

The exact etiology is not known. There are two possible theories:

I- Congenital theory

Claims that congenital dermal inclusions (overlying the lower sacral region) get infected and present after puberty.

II- Acquired theory (more acceptable)

- assumes that loose hairs from the head or back gravitate to the skin over the sacrum and coccyx.

- In the presence of pressure from sitting, a rolling movement of the buttocks, sweat & poor hygiene → the hairs are drawn through skin to accumulate in the subcutaneous tissue with debris & organisms.

The following facts favour the acquired theory:

1. Pilonidal sinus may occur in other areas as:
 - a) In the umbilicus
 - b) In the webs of fingers of barbers.
2. Microscopic examination shows only loose hairs in the cavity but there are no hair follicles.
3. The tip of the hair is always directed inward.
4. Liability to repeated recurrences

Pathology

- There is a cavity:

- Content → loose hairs

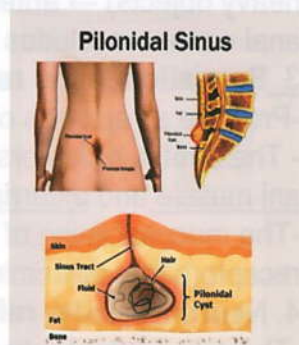
- Lining → granulation tissue

- Site → in the subcutaneous space overlying the lower sacrum.

- Opening → on the skin in the middle line by one or more openings (their tracks being partially epithelialized)

Pilonidal sinus is not an anal disease but is described in this chapter because of its closeness & possible misdiagnosis as an anal fistula.

- It is a common minor condition of the skin overlying the sacrum



-Intermittently, aerobes & anaerobes proliferate → an abscess which may empty through the midline openings or point and open laterally and inferiorly → 2^{ry} sinuses.

-Abscess formation has a tendency to recurrence.

Clinical picture

Type of patient

Typically affects young adult males with dense, dark, strong hair.

Females are also affected but to a lesser extent.

Onset: seldom develops after the age of 30 years.

Symptoms:

1. May be asymptomatic.
2. Local discomfort & discharge (more usually)
3. Acute abscess formation

Signs:

-Discharging midline and lateral openings, sometimes with loose hairs protruding out of them

Differential diagnosis

1. Perianal abscess.
2. Anal fistula.

Treatment

I-Pilonidal abscess

- 1-By incision and drainage of pus.
 - 2- Loose hairs are removed
 - 3-The wound is left open & packed to heal by granulation tissue
- If a sinus recurs → treated on an elective basis.

II-Pilonidal sinus

Different options are available:

1- Laying out the cavity & side tracks and curettage.

- Phenol cauterization may be applied to the tracks.
- The resulting wound is left open & packed to heal by 2^{ry} intention

2- Localized excision of the cavity and side tracks.

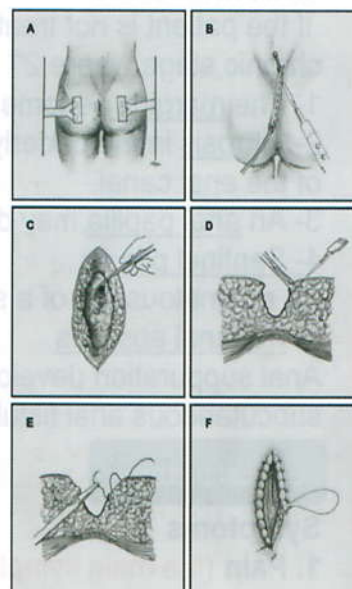
- The wound may be left open to granulate or is closed by sutures to accelerate healing

After any of the above operations, in order to reduce the possibility of recurrence the patient is advised to:

- 1-keep the area clean
- 2- Shave it regularly



- Pilonidal disease tends to recur if improperly treated
-Wide wedge excision down to the sacrum no longer practiced as it is considered an unnecessarily extensive operation with very long healing time



Anal Fissure

Definition

An elongated ulcer which occurs in the lower half of the anal canal

Etiology

1-Constipation (commonest cause)

Passage of a hard faecal bolus → hits the posterior wall of the anal canal below the dentate line (the epithelial lining of this area is less supported by muscular tissue) → stretched and gives way.

2- Repeated deliveries

Relaxation or tearing of the perineum during delivery → lack of support to the anal lining

3-Crohn's disease (Rare)

Pathology

Site → always in the midline

1-in the midline posteriorly (90%)

2- in midline anteriorly (10%)

Acute fissure

- A superficial tear in the lower ½ of the anal canal in the midline posteriorly.

- Pain → persistent spasm of internal sphincter → prevents healing.

Chronic fissure

If the patient is not treated efficiently, the fissure will proceed to the chronic stage where 2nd pathological changes occur:

1- The margins become indurated.

2- Fibrosis in the underlying internal sphincter muscle → narrowing of the anal canal.

3- An anal papilla may develop at the upper end of the fissure.

4- Sentinel pile

An edematous tag of a skin may form at the lower end of the fissure

5- Perianal abscess

Anal suppuration develop in relation to the fissure → burst → subcutaneous anal fistula.

Clinical picture

Symptoms

1. Pain (the main symptom)

-Character: sharp, agonizing

-Onset: starts during defecation

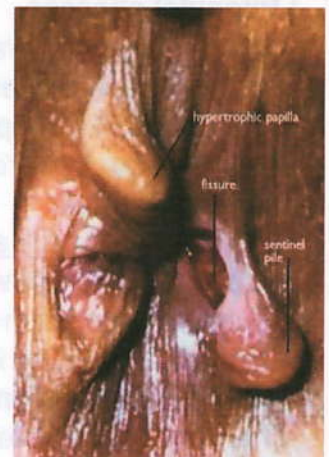
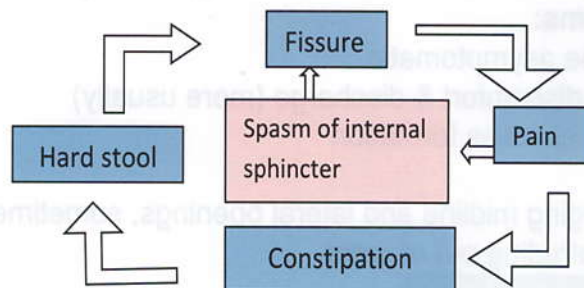
-Duration: continue for a few hours after defecation

- Course: the patient gets some relief until the next defecation.

In subacute cases → patient get some remission for a few days or weeks but usually the pain recurs.

2. Constipation.

The severe pain of the fissure → make patient postpone defecation → more constipation (a vicious circle)



3. Bleeding

Only a slight streak of blood on the surface of the stools (compared with haemorrhoids)

4. Anal discharge

- Slight

-If abscess forms & bursts → purulent discharge.

5. Reflex symptoms (common)

a) Burning micturition b) dysmenorrhoea c) pain along the thighs

Signs

I- In acute anal fissure

Inspection:

1- The anal verge reveals a tightly contracted, puckered anus.

2- If the 2 gluteal folds are gently pulled laterally → a small tear seen in the midline posteriorly

DRE:

-It is very painful and inadvisable to do it

II- In chronic fissure

Inspection

1-fissure is seen

2-An anal papilla or a sentinel pile may be present

Palpation → indurated edges is felt

(Done after application of an anaesthetic gel for a few minutes)



Any patient with atypical presentation of anal fissure → histopathological examination of the excised specimen should be performed.

- D.D. of painful anal conditions:

1. Anal fissure.
2. Perianal suppuration.
3. Prolapsed strangulated piles
4. Acute perianal haematoma.
5. Carcinoma of the anus.
6. Proctalgia fugax (idiopathic)

Differential diagnosis

1. Early carcinoma of the anus.
 2. Fissures due to Crohn's disease.
- Usually multiple, occur at any site, not very painful & not indurated.
3. Tuberculous ulcers (have undermined, cyanotic edges)
 4. Anal chancre (presents as a painful ulcer)

Treatment

A-Acute fissure

(Essentially medical but surgery is sometimes needed)

The aim: relieve the persistent spasm of the internal sphincter → the fissure can heal.

Treatment

(i) life style changes

1. High-fibre diet
2. Some laxatives
3. Frequent warm water baths → a sedative effect.

Ensure bulky soft stools

(ii) Medical

1. A local anaesthetic ointment as **2% or 5%** lignocaine
2. Glyceryl trinitrate ointment **0.2%** or bethanecol ointment **1%**
3. Suitable doses of analgesics are prescribed.

(iii) **Surgical** (If all the previous measures fail to relieve pain within a few days)

-Do lateral internal sphincterotomy under general anaesthesia

B-Chronic fissure (Treatment is surgical)

(i) If the fissure is not very chronic:
lateral internal sphincterotomy operation

-Is very successful.

-The internal sphincter is divided at the 3 o'clock position.

-Relief of the spasm of the internal sphincter → allow healing.

-The operation can be done by the closed or open method.

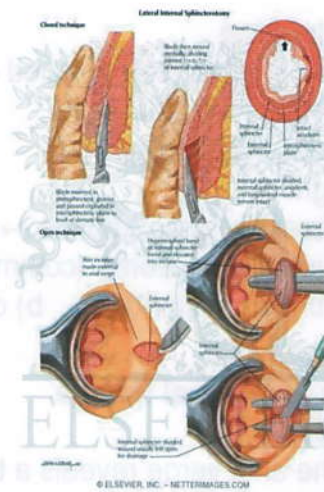
(ii) If the fissure is heavily fibrosed with a sentinel pile:

Fissurectomy and posterior internal sphincterotomy

- A triangular segment with its apex upwards (including the fissure, anal papilla and sentinel pile) is excised

-The excision includes the fibrosed part of the internal sphincter muscle as well.

(iii) Local injection of Botulinum toxin (Botox) (A recent line)



Haemorrhoids (piles)

-In Greek, haima means blood while rhoos means flowing.

- In Latin, pila mean a ball.

-So the accurate terminology should be:

a) Haemorrhoids if the main complaint is bleeding per rectum

b) Piles if the main problem is prolapse



Anatomy

Anal cushions

-A vascular plexus beneath the epithelial lining of the anal canal formed terminal branches of the superior haemorrhoidal vessels

- are usually arranged at the 3,7,11 o'clock positions around the circumference of anal canal.

- play an important role in the normal continence mechanism.

-Their congestion, enlargement & prolapse → haemorrhoids

Types

1- Internal haemorrhoids

-Lies above the dentate line

- Covered by mucous membrane & are bright red or purple in color.

2- External haemorrhoids

-Lies below the dentate line

- Are covered by skin.

3- **Interno-external haemorrhoids** → develop if both are present

Etiology

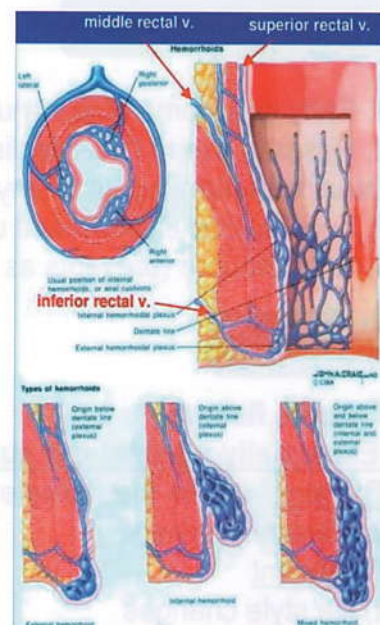
A- Primary haemorrhoids (most common)

No definite cause, predisposing factors are suggested:

1. Genetic factors

-Are more common in certain families

- Congenital weak mesenchyme may have a role.



2. Chronic straining at defecation.

3. Anatomical factors.

Venous drainage of the anal cushions may contribute as follows:

- The anal canal cushions lie unsupported in the loose submucous layer → degeneration with aging of the supporting elastic tissue → engorgement and dilatation → haemorrhoids.
- The tributaries of the anal cushions penetrate the muscular wall of the rectum before joining the superior rectal veins → compressed during defecation (especially in prolonged straining)
- There are no valves in the portal circulation → high hydrostatic pressure at the haemorrhoidal plexus

B- Secondary haemorrhoids (less frequent)

Has definite cause as:

1. Pregnancy

-Due to raised intra-abdominal pressure & relaxing effect of progesterone.

2. Pelvic tumours

-Particularly carcinoma of the rectum → obstruct tributaries of the superior rectal vein.

Rectal carcinoma should be excluded in patients above the age of 40 with bleeding per rectum even if there are evident haemorrhoids.

Clinical picture

Symptoms

1. Bleeding per rectum (the most common complaint)

-Haemorrhoidal bleeding is characterized by:

- Occurs with straining
- Usually at the end of defecation.
- Fresh bright red.
- Jet or drops that are separate from stools.

2. Prolapse

-The vascular cushions enlarge & descend below the dentate line.

-Piles are classified according to their degree of prolapse into:

a) 1st degree piles

- There is no prolapse of piles.
- May have no symptoms or may present by bleeding only.
- Diagnosed only by proctoscopy.

b) 2nd degree piles

- The piles prolapse only during defecation but they are spontaneously reduced at the end of the act.

c) 3rd degree piles

- There is prolapse of piles during defecation and the patient has to manually reduce them.

d) 4th degree piles

There is permanent prolapse of piles.

3. Anal discharge and pruritus (itching).

-In piles that prolapse beyond the anal verge → mucous discharge → pruritus ani.

4. Pain and discomfort

- Indicates either a complication of the hemorrhoid or the presence of another problem as a fissure.

1st Degree: No Prolapse. Just prominent blood vessels



2nd Degree: Prolapse upon bearing down but spontaneously reduced.



3rd Degree: Prolapse upon bearing down and requires manual reduction.



4th Degree: Prolapsed and cannot be manually reduced.



Uncomplicated haemorrhoids are painless.

Signs

Inspection:

1. **In early cases** → no abnormality can be seen outside the anal verge (only proctoscopy can reveal the presence of internal haemorrhoids → bluish bulges below the anorectal ring)

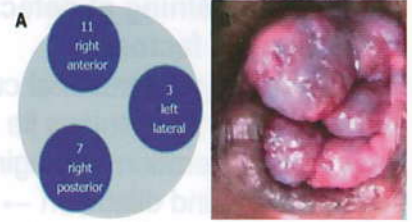
2. **In late cases** → prolapsing piles can be seen in one or more of the three primary positions → **mother piles**.

- There may be daughter piles between the main 3 ones.

DRE:

- Uncomplicated piles are impalpable because are compressible.

- Is essential to exclude a palpable rectal cancer.



Sigmoidoscopy should be performed to exclude rectal cancer

Complications

1. Profuse haemorrhage

2. Anaemia.

3. Strangulation

- when one or more of the internal haemorrhoids prolapse → gripped by the external sphincter → interference of the venous return → the haemorrhoids become very tense, swollen and very painful.

4. Thrombosis

- If strangulation is not relieved rapidly → thrombosis & the affected hemorrhoid becomes dark purple or black

5. Ulceration

May follow strangulation and thrombosis.

6. Gangrene

If the strangulation is very tight → the arterial supply is constricted → superficial slough or the whole hemorrhoid sloughs → an ulcer.

7. Suppuration

- Is the result of infection of a thrombosed hemorrhoid

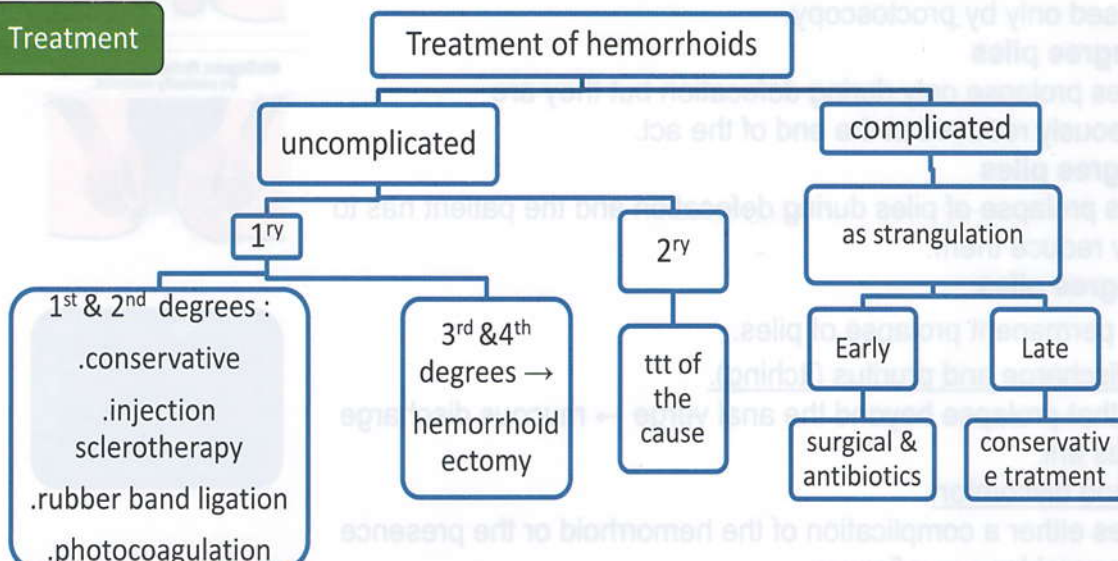
8. Portal pyaemia

Is a rare complication of infected haemorrhoids



It is said that strangulated haemorrhoids had cost Napoleon Waterloo battle and the French their empire

Treatment



A-Primary haemorrhoids

I- For first and second degrees

1. conservative treatment:

Indication: → early cases.

Method:

- High fibre diet.
- Small doses of laxatives.
- Avoidance of straining at defecation.
- Suppositories that contain decongestants

2. Injection sclerotherapy

-Indications: Bleeding first and second degree hemorrhoids.

-The idea

To inject an irritant material in the submucosa at the pedicle of the haemorrhoids → fibrosis → obliterate the venous plexus & pull the prolapsed cushions up.

-Method

- Does not need anaesthesia (performed in a non sensitive area)
 - 3 ml of 5% phenol in almond oil is injected at the upper end of each hemorrhoid in the submucosa (not in the vessels).
- The three haemorrhoids can be injected at the same time.
-The procedure can be repeated once or twice after 2 weeks.

- Complications

1. Severe pain

If the injection is done at a low position in the sensitive anoderm

2. Necrosis of the overlying mucosa: If the injection is superficial.

3. Hematuria: If the injection is deep it.

4. Allergic manifestations.

5. Submucous abscess formation

3. Rubber band ligation

-Indications: in second and early third degree haemorrhoids.

-The idea: place a tight elastic rubber band around the pedicle of the hemorrhoid → ischemic necrosis and separation

- Does not need anaesthesia

4. Infrared Photocoagulation

-Indications: Bleeding first and second degree hemorrhoids

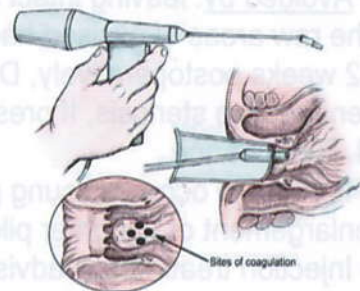
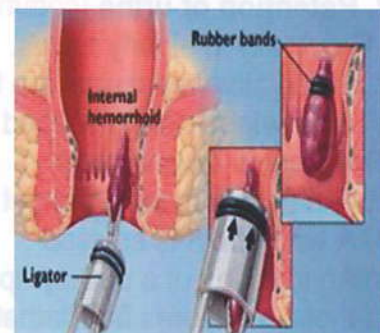
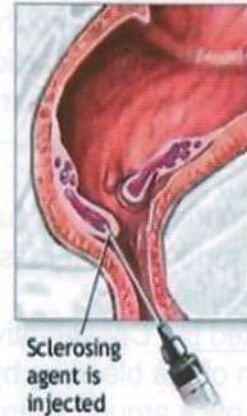
- The idea: produce a tissue temperature of 100°C → coagulative necrosis → dead tissues separate after 10-14 days → a granulation lined ulcer.

-This is an effective and painless method.

II- For third and fourth degree

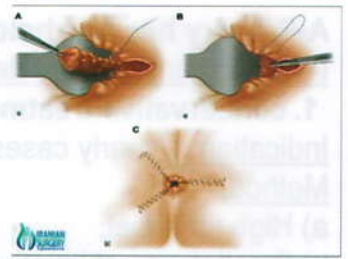
Surgery (**Hemorrhoidectomy**) is recommended

-The idea: to excise the hypertrophied vascular cushions with the overlying redundant skin.



-Method:

1. General or spinal anesthesia.
2. The patient is placed in the lithotomy position.
3. An Allis forceps is applied to the skin overlying each external haemorrhoids and an artery forceps on each internal haemorrhoids.
4. Traction is applied on both the Allis and artery forceps & a V shaped cut is made in the skin overlying the external hemorrhoid.
5. Dissection exposes the lower end of the internal sphincter.
6. Pedicle of internal hemorrhoid is ligated by strong vicryl suture.
7. Each hemorrhoid is dealt with in the same manner then excised 1 cm distal to the ligature.
8. Leave intact skin and mucosa between the individual pedicles.
9. Postoperative strong analgesics.
10. Some surgeons nowadays perform stapled Haemorrhoidectomy



-Complications:

1. Haemorrhage (reactionary or 2^{ry})

(i) **Reactionary haemorrhage** is treated by:

- a) Giving morphia injection
 - b) Applying local pressure by a piece of gauze.
- If the bleeding persist → surgical ligation of the bleeding point is.

(ii) **Secondary haemorrhage**

-more serious as there is 2^{ry} infection & the tissues are very friable.

- Treated by: Conservative measures, if fail → surgical ligation of the bleeding by under running sutures, if fail to → a pack around a large rectal tube is inserted in the anal canal for 2-3 days.

2. Retention of urine (common)

- Especially in elderly males.
- Conservative measures are tried at fist:
 - a) An analgesic is prescribed for the pain
 - b) If there is an anal pack → remove
 - c) The patient is asked to get out of bed
 - d) A warm bath is advised
 - e) An injection of a parasympathomimetic drug as carbachol
- If these measures fail, catheterization is advised.

3. Anal stricture

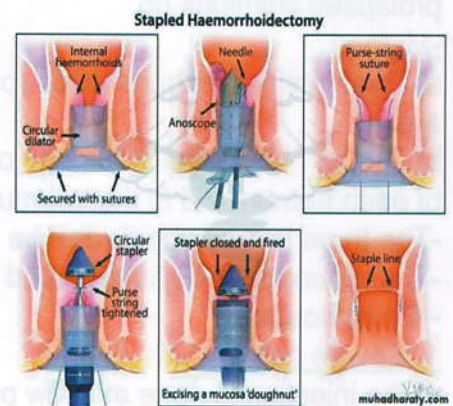
- Avoided by: leaving intact areas of anal skin and mucosa between the raw areas of excised haemorrhoids.
- 2 weeks postoperatively, DRE is performed to check that there is no tendency to stenosis, If present → gradual anal dilation is started.

4. Recurrence

- Is liable to occur in young patients and is usually due to enlargement of daughter piles.
- Injection treatment is advised.

5. Fissure

- One of the cutaneous wounds not heal completely → fissure



Presentations of common anal conditions

1. Fissure:

Pain with & after defecation

2. Uncomplicated piles:

Bleeding & Prolapse

3. Strangulated piles:

Pain and prolapse

4. Abscess:

Pain (persistent) and fever

5. Fistula → Discharge

- Treatment is conservative ,If it fails → internal sphincterotomy

B- Secondary haemorrhoids → treatment of the cause

Treatment of prolapsed strangulated haemorrhoids:

A- Early cases → surgical intervention under antibiotics

B- Delayed cases

-the tissues are friable & there may be 2nd infection, therefore → conservative measures:

1. Rest in bed.
2. Antibiotics.
3. Analgesics.
4. Frequent warm baths.
5. Decongestive ointments & local compresses by lead subacetate lotion

The anal glands

-site: in a plane between the internal and external anal sphincters & communicate by their ducts to the anal mucosa at the level of the dentate line

Acute perianal haematoma (thrombosed external hemorrhoid)

Etiology

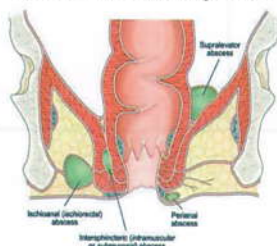
A very painful condition due to rupture of a dilated anal vein 2nd to straining at defecation, coughing or lifting a heavy weight → haemorrhage in the loose subcutaneous connective tissue (usually in the lateral position)

Clinical picture

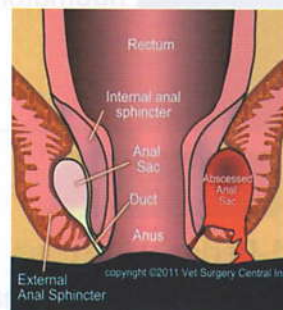
A tense, tender, bluish swelling covered by smooth shining skin

Fate

- Untreated → the haematoma usually resolves.
- May suppurate, ulcerate through skin with extrusion of the blood clot fibrosis → a cutaneous tag



or



Treatment

Early cases with severe pain → evacuate the haematoma

Anorectal Abscess

Etiology

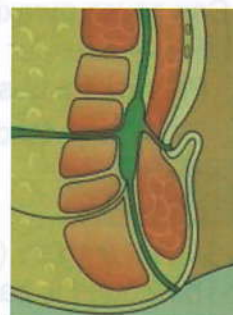
A-Primary anorectal abscess

Is due to infection of the anal glands or the skin glands by Gram negative bacilli → an inter-sphincteric abscess which may spread:

1. Downwards → Perianal abscess
2. Outwards → Ischiorectal abscess
3. Inwards → submucous abscess

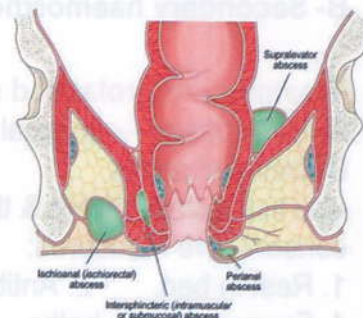
- In the majority of these abscesses, there is an inner opening in the anal canal (drainage of the abscess → fistula)

- Infection of the apocrine glands or hair follicles of the perianal skin → perianal suppuration in 15-25% of cases (the causative organisms are usually Staphylococcal) & there is no communication with the anal canal.



B-Secondary anorectal abscess

1. Inflammatory bowel disease as : Crohn's disease & ulcerative colitis.
2. Specific infections as tuberculosis.
3. Anorectal carcinoma.
4. Infection of a perianal haematoma, thrombosed haemorrhoids or anal fissure (fissure abscess).



Classification and Etiology

	Perianal abscess	Ischio-rectal abscess	Submucous abscess	Pelvi-rectal abscess
Incidence	60 %	30 %	5 %	5 %
Site	subcutaneously adjacent to the anal orifice	ischio-rectal space	in the submucous space <i>above</i> the dentate line	between the upper surface of the levator ani & pelvic peritoneum
Etiology	downward spread of an intersphincteric abscess or infection of a perianal haematoma	to lateral extension of an intersphincteric abscess	1. Inward spread of intrasphincteric abscess 2. Infected piles after injection	1. This is actually a pelvic abscess 2 nd to appendicitis, salpingitis or diverticulitis 2. injury of the levator ani muscle by an instrument during drainage of an ischio-rectal abscess (rare)

Clinical picture

1-Perianal abscess

Pain and constitutional symptoms (not marked)

2. Ischio-rectal abscess

- High fever with marked constitutional manifestations.
- A large, indurated, tender swelling filling the ischio-rectal space.
- There is throbbing pain and pitting edema.

3. Submucous abscess

- Severe pain & fever but nothing is seen outside the anal verge.
- Can cause pyrexia of unknown origin.
- DRE → a tender boggy swelling.

4. Pelvirectal abscess

DRE → tender boggy swelling

Treatment

- 1-Urgent surgery (incision and drainage) under general anaesthesia.
- 2-The wound is packed with daily dressings.
- 3- If the abscess is near the anal verge → look for an internal fistulous opening (if present → treat the patient as if having a fistula)

-If ischio-rectal abscess is not drained early → extend through the Postsphincteric space → involve the other side → **horse shoe abscess.**

-The surgeon should not wait for fluctuation to drain this abscess

- The patient who had drainage of an anorectal abscess should be warned that he may develop an anal fistula

Anal Fistula (Fistula in Ano)

Definition

A track lined by granulation tissue that extends:

From → an opening at the skin of the perianal region

To → the cavity of the anal canal or rectum

Etiology

Most fistulae arise from a pyogenic intersphincteric abscess which has been followed by: spontaneous rupture or inadequate drainage

Pathology

Factors that lead to persistence of anal fistulae

1. The anal glands act as a reservoir for infection.
2. The presence of an internal opening → recurrent activation of infection → build-up of pus → discharges internally & externally.
3. Underlying specific diseases may be associated with persistent fistulae such as: Crohn's disease, tuberculosis & ulcerative colitis.
4. Faecal material may act as a foreign body
(It is rare to find such material on exploring the fistulous track)

Classification

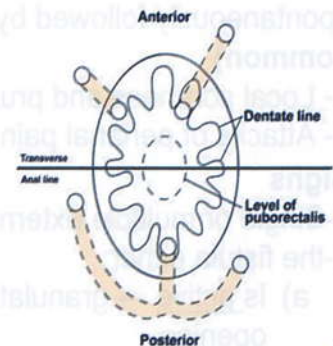
The outline of the fistulous track is considered in two planes:

A-The horizontal plane → by **Goodsall's** rule

- States that the position of the internal opening in the circumference of the anal canal depends upon the relationship of the external opening to an imaginary line drawn between the ischial tuberosities bisecting the anus.

1-If external openings are anterior to Goodsall's line → internal openings are on the same radius having short direct fistulous track

2- If external openings behind this line → tracks communicate with an internal opening in the midline posteriorly



B- The vertical plane

- It is important to determine the vertical axis of fistulous track to avoid injury of the sphincteric control of continence during surgery.

I- Standard classification

According to position of internal opening in relation to the anorectal

ring: 1- **High fistula**: above anorectal ring

2- **Low fistula** : below anorectal ring

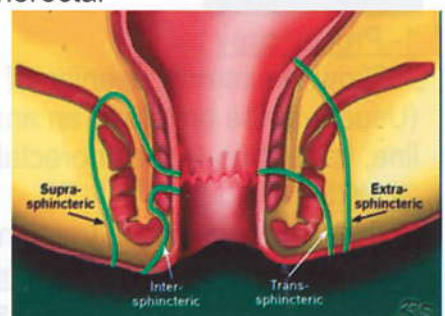
II-New classification (**Park's** classification)

According to the course & relation of the fistulous track to the anorectal sphincter muscles.

1-**Intersphincteric fistula**

-Begins at the dentate line

-Extends to the perianal skin, traversing the plane between the internal & external anal sphincters.



-Sometimes, there are multiple external openings but usually there is a single internal opening

- Almost all anal fistulae have their internal opening at the level of the dentate line

2- Transsphincteric fistula

- Begins at the dentate line
- Traverses both internal & external anal sphincters
- Opening through the ischiorectal fossa in the perianal skin.
- May be complicated by additional blind tracks.

3- Suprasphincteric fistula

- Begins at the dentate line
- communicates with an intersphincteric abscess cavity then traverses the entire thickness of the external sphincter and puborectalis muscle (hence supra-sphincteric).
- Open in perianal skin after downward passage across the ischiorectal fossa

4- Extrasphincteric (supralelevator) fistula

- Opening: in the rectal wall
- Traverses the ischiorectal fossa through the levator ani muscle
- Another opening in the perianal skin (their track coursing outside the plane of the external anal sphincter)
- Usually have an intermediate communicating track passing through the external & internal sphincters to the original internal opening at the level of the dentate line of the anal canal.
- These fistulas are usually complex and have several causes such as:
a) High anorectal abscess rupturing into the rectum
b) Rectal trauma c) Rectal Crohn's disease d) pelvic abscess

Clinical picture

Symptoms

- 1- History of a previous perianal abscess which discharged spontaneously followed by intermittent or persistent discharge (**Most common**)
- 2- Local soreness and pruritus ani.
- 3- Attacks of perianal pain (occur as recurrent abscesses build up)

Signs

- 1-Single or multiple external openings next to the anal orifice
- 2-the fistula either:
 - a) Is active → granulation tissue & pus may be seen at the opening.
 - b) Healed temporarily.
- 3- The perianal skin shows induration.
- 4- DRE → an internal opening can sometimes be felt.

Investigations

1- Proctoscopy

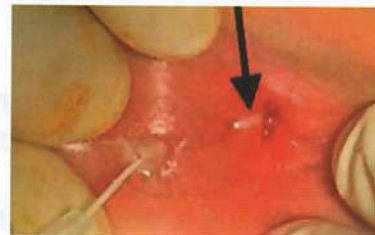
- Show the internal opening of the fistula
(Usually at the bottom of an anal crypt at the level of the dentate line, Its relation to the anorectal ring must be clearly defined)

2- MRI

Will visualize the fistula tract and its relation to the anal sphincters

3-colonoscopy and/or barium enema

Are rarely needed if Crohn's disease is suspected



Treatment

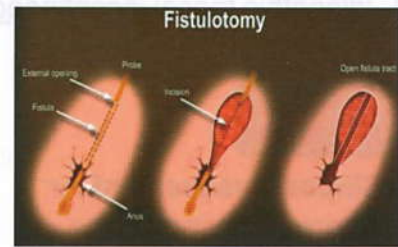
-**Aim:** eradication of sepsis with preservation of anorectal function.

-**Method:**

A-Fistulotomy

- 1- The fistula track is laid open
- 2- The track is curetted
- 3- The edges are trimmed
- 4- Leave an open well-drained wound that heals by 2^{ry} intention.

According to type of fistula



Type of fistula	intersphincteric fistula	trans-sphincteric fistula	supra-sphincteric fistula		Extrasphincteric fistula
Divided part	part of the internal sphincter	part of internal sphincter & superficial part of external sphincter	either		proximal colostomy + staged fistulotomy+ ttt of underlying cause
			Single stage Fistulotomy →entire sphincter anal mechanism	Staged fistulotomy	
Effect on continence	no disturbance	Some minor temporary disturbance	surely produce incontinence	Better continence	ttt is complicated and complex

A staged fistulotomy

The initial stage

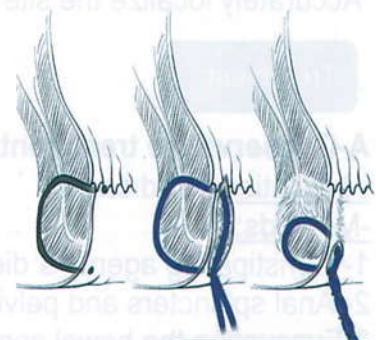
- 1-Deroof the lower part of the intersphincteric component of the fistulous track by dividing the internal sphincter up to the internal opening at the dentate line.
- 2- A Seton suture is left in place through the rest of the track, tightly tied around the puborectalis & external sphincter.

Two weeks later

The rest of the track is laid open guided by the Seton suture.

B- Fistulectomy

Is complete excision of the fistulous track.



In staged fistulotomy, the fibrosis prevents adherent ends of the sphincter from retraction widely apart → maintaining continence.

Faecal Incontinence

Definition

Inability to retain the rectal contents at will

Etiology

1. Damage to the anal sphincter mechanism by:

- a) Obstetrical trauma → complete perineal tear.
- b) Surgical trauma as during surgery for a high anal fistula.
- c) Accidental trauma.

2. Complete rectal prolapse

The prolapsing rectum → stretches the anal sphincters → damage

3. Neurological diseases

- a) Trauma or tumours affecting the 2nd, 3rd & 4th sacral nerves.
- b) Diabetic autonomic neuropathy.

4. Idiopathic faecal incontinence.

Assessment

A-Clinical assessment for:

- 1- Degree of incontinence
(To flatus only, to flatus & fluid stools or to solid stools as well)
- 2- History of trauma, surgery or difficult labour.
- 3- Rectal prolapse.
- 4- Sphincter contraction.
- 5- Neurological assessment.

B-Investigations

1. Manometry

Detect rectal function, length of the anal sphincters & their strength.

2. Electromyography: assesses the function of the pudendal nerve.

3. Defaecography: to assess the anorectal angle.

4. Trans-anal ultrasound and MRI

Accurately localize the site of sphincteric damage.

Treatment

A-Conservative treatment

- Indication: mild cases.

- Methods:

- 1- Constipating agents & dietary manipulations to thicken the stools.
- 2- Anal sphincters and pelvic floor exercises (**Biofeed back**).
- 3- Evacuating the bowel completely with a glycerine suppository in the morning → avoid soiling later in the day.

B-Surgery

1- Repair of divided sphincters.

2- Rectopexy

Restore continence to **50%** of those having complete rectal prolapse.

3- Postanal repair

- Indication: idiopathic incontinence.

- The aim: restore the right angle of the anorectal junction.

- The levator ani & sphincters are plicated behind the anorectal junction → pushing it forwards.

- Excessive descent of the perineum with straining is a common accompanying feature with rectal prolapse → pudendal nerve traction neuropathy.
- Diarrhoea accentuates incontinence & may even produce it in patients with borderline weak sphincters

If the anal sphincters is severely disrupted → the gracilis muscle is mobilized to encircle the anal canal → new sphincter.
- A special pacemaker is implanted to transform the muscle from a fast twitch to slow-twitch muscle

Malignant Tumours

Pathology

Types

1- Squamous cell carcinoma (The commonest type)

Arising from the anoderm.

2- Adenocarcinoma (Less common)

risers above the dentate line.

Spread

1- If the tumour is below the dentate line → inguinal lymph nodes

2- If the tumour is above the dentate line → para-aortic lymph nodes

Etiology

Infection with human papilloma virus (important factor)

Clinical picture

Symptoms

The patient presents by a lump, pain or discharge.

Signs

1- DRE → an indurated ulcer that bleeds easily

2- Inguinal lymph nodes are examined for metastases.

Treatment

A-Carcinoma of the anal verge

1- Wide local excision with 2.5 cm safety margin.

2- If there are metastatic LNs → inguinal block dissection

B-Carcinoma of the anal canal

One of two lines may be performed.

1- Chemoradiation.

- A course of combination chemotherapy is followed by radiotherapy (Squamous cell carcinoma is radiosensitive).

- The patient is examined after **4-6 weeks**, if there is any residual tumour → abdominoperineal resection

2- Abdominoperineal resection

- of the anal canal and rectum with a terminal colostomy

- Indication: large tumours.



Biopsy is essential before institution of treatment

Pediatric Surgery

DERMOID CYSTS

Definition

A congenital inclusion cyst of skin & skin appendages lined by stratified squamous epithelium → desquamation → sebaceous cheesy material (inside the cyst)

Site

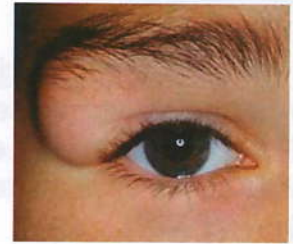
1. On the scalp & eyebrows (commonly)
2. In the midline of the nose, neck & upper chest

Clinical picture

- 1-Painless swellings
(Completely mobile or fixed to the skin and deeper structures)
- 2- Dermoid cysts of the eyebrows and scalp → depression in the underlying bone → a smooth, punched-out defect on radiographs of the outer table of the skull (do not extend intracranially)

Treatment

- 1-They should be excised intact
(Incomplete removal → recurrence)
- 2-Those arising near the eyebrows should be excised through an incision adjacent to the hairline.
-The eyebrows should not be shaved nor should the incision go through any eyebrow follicles, if so → permanent area without hair
- 3-There are reports of removing facial dermoids using an endoscope via an incision in the scalp→ avoiding a facial scar



Cysts of the face & scalp that are located in midline can extend intracranially so *it is essential to obtain an MRI or CT scan preoperatively.*
- Dermoid cysts of the midline neck may be confused with thyroglossal duct cysts → dermoids do not move with swallowing or tongue protrusion (as they are not deep to the strap muscles unlike thyroglossal cysts)

BRANCHIAL ANOMALIES

- During the 1st month of fetal life, the primitive neck develops **4** external clefts & **4** pharyngeal pouches separated by a membrane.
- Between the clefts & pouches are branchial arches.
- The dorsal portion of the 1st cleft becomes the external auditory canal; the other clefts are obliterated.
- The pharyngeal pouches persist as adult organs.
 - 1st pouch → auditory tube, middle ear cavity & mastoid air cells.
 - 2nd pouch → incompletely regresses & becomes the palatine tonsil and the supratonsillar fossa.
 - 3rd pouch → the inferior parathyroid gland and the thymus
 - 4th pouch → forms the superior parathyroid glands.
- Fistulas communicating between the anterior border of the sternocleidomastoid muscle & tonsillar fossa are of 2nd branchial origin.
- 2nd branchial cleft abnormalities occur **6** times > 1st cleft anomalies.

-Branchial anomalies are remnants of fetal branchial apparatus.
-A tract of branchial origin → a complete fistula
- One end may be obliterated → an external or internal sinus
- Both ends may obliterate → an aggregate of cells → a cyst.

Branchial cyst and fistula

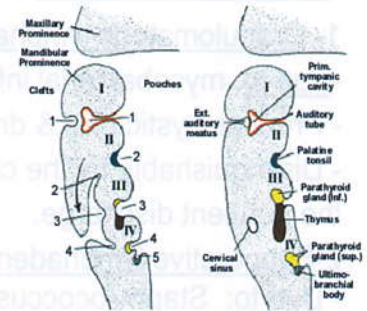
Etiology

Normal development

During embryological development, 2nd branchial arch grows rapidly covering the 3rd & 4th arches then it fuses with the neck.
-The space between 2nd arch & rest of the arches turns into → a cervical sinus which disappears.

Abnormal development

- A branchial fistula develops if 2nd arch doesn't completely fuse with the neck
- If the cervical sinus persists → a branchial cyst.



Pathology

A branchial cyst

- Lining: squamous epithelium
- Content: a clear fluid rich in cholesterol crystals (microscopically identified in aspirated cyst fluid)
- The cyst wall is surrounded by lymphatic tissues → gets infected

A branchial fistula

- Etiology: usually congenital but occasionally 2^{ry} to ruptured infected branchial cyst.
- The track:
 - a) Is lined by → squamous or ciliated epithelium
 - b) Extension → up to the side wall of the nasopharynx (**fossa of Rosenmuller**).
 - c) Course → passes between the bifurcation of the common carotid artery, deep to the posterior belly of digastric muscle and superficial to the hypoglossal nerve

Clinical picture

Branchial cyst

- 1- Usually appears at late childhood.
- 2- Presents as a swelling protruding from beneath the anterior border of upper third of the sternomastoid muscle
- 3- Has a smooth surface, mobile & fluctuant.
- 4- Commonly mistaken for a cold abscess (differentiated by finding cholesterol crystals in the aspirate)



Branchial fistula

- 1- Presents at birth as a pin point opening at the anterior border of lower third of the sternomastoid muscle
- 2- The opening discharges mucoid material but when gets infected → becomes purulent.
- 3- May occasionally be confused with a tuberculous sinus



Differential diagnosis

1- Granulomatous lymphadenitis

- Due to: mycobacterial infections
- Produce cystic LNs & draining sinuses
- Distinguishable by the chronic inflammatory reaction that precedes the purulent discharge.

2- Suppurative lymphadenitis

- Due to: Staphylococcus aureus
- Resemble an infected branchial remnant.
(Lymphadenitis → is curative & completely heals while branchial remnant persist after the infection resolves)

3- Hemangiomas & lymphangiomas

- Are soft, spongy tumor masses
(Branchial cysts have a firmer consistency)
- Lymphangiomas may transilluminate (branchial cysts do not)

4- Carotid body tumors

- Are quite firm & located at the carotid bifurcation
- Occur in older patients.

5- Lymphomas

- Produce firm masses in the area where branchial remnants occur but multiple matted nodes rather than a solitary cystic tumor
- Firm cordlike tract may be palpable along its course.

Nearly all branchial abnormalities should be excised early in life since repeated infection is common → difficult resection

Treatment

A- Branchial cyst

By excision through a transverse neck incision

B- Branchial fistula

- The whole track should be excised → through multiple transverse neck incisions: a) Small one around the fistula opening
b) The other at higher level below the jaw

- A ureteric catheter is introduced into the track to facilitate its identification during surgery

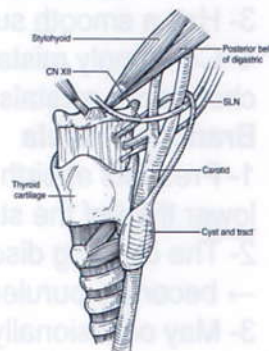
- Infected sinuses and cysts :

- 1) Require initial incision and drainage.
- 2) **6 weeks later** (when the acute inflammation subsides) → staged excision of the tracts

- Ensure complete excision of cyst wall or fistula tract (including skin punctum) since incomplete removal → recurrence & infection

- Take care not to injure the following important surrounding structures:

- a) Nerves: the facial, hypoglossal & glossopharyngeal
- b) The carotid artery
- c) internal jugular vein



LYMPHANGIOMA (CYSTIC HYGROMA)

Definition

Are benign multilobular, multinodular cystic masses lined by endothelial cells

Etiology

Normal development

-The lymphatic system develops by the coalescence of multiple small lymph vesicles → large accumulation present lateral to the jugular vein and is called the jugular lymph sacs.

Abnormal development

If some vesicles of the jugular lymph sac fail to join the lymphatic system they become sequestered and form a cystic hygroma.

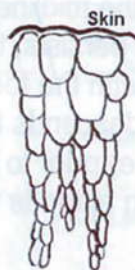
Pathology

Site

- Most commonly in the posterior triangle of the neck (75%)
- In the axilla (20%)
- The remainder in the mediastinum, retroperitoneum, pelvis & groin.

Gross picture

The swelling consists of multiple cysts with the larger ones near the surface while the smaller ones are deep & infiltrating tissue planes.



Microscopic picture

Each cyst is lined by endothelial cells and contains clear lymph.

Clinical picture

Type of patient

50-65% appears at birth & 90% are evident by the 2nd year of life.

Symptoms

- 1-The majority are asymptomatic (a painless swelling)
- 2- Large lesions invading the floor of the mouth → symptoms referable to pharyngeal or upper airway obstruction

Signs

- 1-The swelling appears bluish & translucent to light.
- 2- Soft, partially compressible & ↑ in size with coughing & crying.
- 3-The skin is never involved, although the lesion may be densely adherent to the undersurface of the dermis

Preauricular lesions:

- Anomalous development of the auricle → Preauricular sinuses, cysts & cartilaginous
- Are unrelated to branchial anomalies.
- The sinuses are often short and end blindly.
- They can be cosmetically unappealing and often become infected.
- Superficial skin tags and cartilaginous rests are easily excised without risk to other structures.
- Preauricular sinus tracts may be very deceptive in their extent and extensive dissection has risks to damage branches of facial nerve



Treatment

There are two modes of treatment, the choice depends on imaging studies (CT, MRI):

1-sclerotherapy

-Indications: is most effective for unilocular or macrocystic lesions.

-Method: Intralesional injection of a sclerosing agent as:

a) OK-432 (a lyophilized mixture of *Streptococcus pyogenes* & penicillin G potassium)

b) Bleomycin

c) Doxycycline.

2- Excision

-Method: with bipolar cautery to ensure a hemostatic dissection & decrease the incidence of lymph leak and nerve injury.

- The recurrence rate following surgery is 50%.

Thyroglossal Cyst

Etiology

Normal development

-During the 4th week of gestation, the thyroid gland develops from an invagination in the floor of the primitive pharynx located between the first pair of pharyngeal pouches.

Abnormality

-If the thyroid does not descend normally → mass at the base of the tongue or anywhere in the midline of the neck along path of descent
-If the thyroglossal duct persists, the epithelial tract forms a cyst that usually communicates with the foramen caecum of the tongue.

-The thyroglossal duct descends through 2nd branchial arch (which becomes the hyoid bone) prior to its fusion in the midline → therefore the persistent tract often extends through the hyoid bone.

Pathology

-A midline tubulodermoid cyst arising in thyroglossal duct remnant.

-Site

- At any level from the foramen caecum of tongue to the suprasternal notch,

-Commonly → in the midline just below the hyoid bone
(at the level of the thyroid cartilage → displaced usually to the left)

Clinical picture

Type of patient

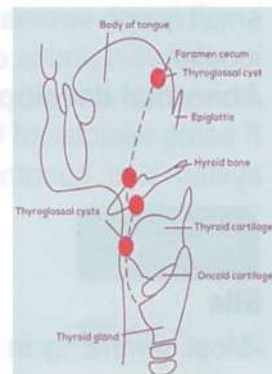
-At any age but is most common during childhood.

Symptoms

-A painless cystic swelling in midline of neck

Signs

- Cystic midline mass that moves up and down on swallowing (since they are deep to the cervical strap muscles) and on protrusion of the tongue (due its relation to the hyoid bone).



The thyroglossal duct can assume a variable relationship to the hyoid bone: Behind, in front or through the body of the bone

- Carcinoma develops more frequently in ectopic thyroid tissue than in normal thyroid glands.



- There may be a palpable track extending from the hyoid bone upwards towards the tongue
- The fluid in the cyst is usually under pressure → give the impression of being a solid tumor.

Differential diagnosis:

- 1-Lymph nodes
- 2-Dermoid cysts → not move with swallowing
- 3- Enlarged prelaryngeal LNs containing tumor metastases.
- 4-lingual thyroid

Complications

- 1-There is a malignant potential of the dysgenetic thyroid tissue located in a thyroglossal duct cyst
- 2- Excision of an ectopic thyroid → remove all thyroid tissue → hypothyroidism.
- 3- **Thyroglossal fistula**
 - The track is lined by columnar epithelium
 - Etiology:** 1-The wall of the cyst is rich in lymphatics that communicate with the cervical lymph nodes → infection → spontaneous rupture or incision & drainage → fistula
 - 2- Inadequate removal of the cyst.

-Clinical picture:

- 1- The opening of the fistula is near the midline on the left side with a crescentic fold.
- 2-discharges mucus & presents with recurrent inflammation



Treatment

- Complete excision is indicated because of the risk of infection and the possibility of the development of papillary carcinoma later in life.
- Acute infection in thyroglossal tracts should be treated with local heat and antibiotics.
- After inflammation subsides (**≈ 6 weeks**) → incision & drainage
- method: **Sistrunk procedure** → remove:
 - 1- Thyroglossal cyst
 - 2-Its epithelial tract
 - 3-The mid portion of the hyoid bone should be removed en bloc with the thyroglossal tract to the base of the tongue
- When the hyoid is not removed → Recurrences



Lingual thyroid

Etiology

Failure of descent of the thyroid

Clinical picture

Tongue swelling with obstructive manifestations as:
Dysphagia, dysphonia, dyspnea, pain or hemorrhage.



Differential diagnosis

Lingual thyroids may be confused with a:

- 1-Hypertrophied lingual tonsil
- 2- Ranula
- 3- Fibroma, angioma, sarcoma, or carcinoma of the tongue.
- 4- Thyroglossal cysts

Distinguished from aberrantly located thyroid glands by needle aspiration or radioiodine scintiscan

Investigations

Thyroid scans → absence of thyroid tissue in normal site in the neck.

Treatment

- 1-Full replacement with L-thyroxine → reduction in size
- 2- Surgical extirpation
- 3-Radioactive-iodine ablation

Sternomastoid tumour and congenital torticollis

Clinical picture

- 1- Presents at birth or 2nd - 6th weeks of life (more common)
- 2-Presents with a hard, nontender, fibrotic mass within the sternomastoid muscle.
- 3- the muscle is shortened → the mastoid process on the involved side is pulled down toward the clavicle & manubrium → the head is abducted to the ipsilateral side and rotated to the contralateral side (toward the opposite shoulder)
- 3-The shoulder on the affected side is raised, and there may be cervical and thoracic scoliosis.
- 4-Passive rotation of the head to the involved side → Resistance & limited movement to varying degrees & muscle appear as protuberant band

Differential diagnosis

From **'wry neck'**

- It is fibrositis causing spasm of sternomastoid muscle (Temporary lasting for a day or two)
- Responds to anti-inflammatory drugs.

Treatment

- 1- With active range of motion exercises:
 - The child's shoulders are held flat to a table and the head is tilted and rotated in a full range of motion.
 - Performed at least 4 times a day for 2-3 months.
- 2- Surgery (rarely necessary)
 - **Indication**: If the muscle continues to become progressively shortened with facial and occipital skull deformity
 - **Method**: both heads of the sternocleidomastoid muscle should be divided through a small transverse incision just above the clavicle.
 - This procedure does not reverse the bony changes that have already developed but prevents progression of the process.
 - Recurrence is rare.

Normal development

The sternomastoid muscle develops through the union of three somites each with its own blood supply.

Abnormal development

Interruption of the blood supply to the central somite (either before or at birth) → muscle infarction → becomes swollen (Called sternomastoid tumour) → fibrous tissue will replace infarcted part → contraction causing congenital torticollis

"Right" sided Torticollis



CONGENITAL DIAPHRAGMATIC HERNIA

Incidence

1 in 2000 live births

It is a highly lethal or morbid disease

Embryology

Normal development

- Fusion of the transverse septum and pleuroperitoneal folds during the 8th week of embryonic development
- Intestine returns to abdomen for rotation & fixation at 10th week

Abnormality

- If diaphragmatic formation is incomplete (embryologic fusion defect) → the pleuroperitoneal hiatus (**foramen of Bochdalek**) persists → herniation of intraabdominal contents into the chest → pulmonary hypoplasia & hypertension and cardiac dysfunction.
- Intestinal non rotation is common as the bowel herniate into the thorax rather than undergoing its normal sequence of rotation and fixation.

Types

1-The foramen of Morgagni (only 2%)

- Occurs at the junction of the septum transversum and the anterior thoracic wall (central diaphragmatic defect)
- It may be parasternal, retrosternal, or bilateral.

2- Bochdalek hernias (commonest)

- Defect is posterior
- Children are asymptomatic & defect is discovered accidentally later in life on a chest radiograph taken for another unrelated reasons

Clinical picture

Symptoms

- Large diaphragmatic defects → symptomatic in the delivery room with tachypnea, grunting respirations, retractions & cyanosis
- Smaller defects → not symptomatic until several days or months.

Signs

1-Abdomen: is scaphoid
(As most of the abdominal viscera is in the hemithorax)

2- The chest

- On the side of the hernia → dull to percussion (but bowel sounds are not usually appreciated)
- When the hernia is on the left → heart sounds may be heard best on the right side of the chest

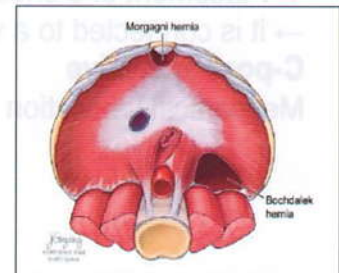
Investigations

A chest x-ray may demonstrate the following:

- A minimal amount of gas within the abdomen

LT side of diaphragm is affected **4-5 times** as frequently as the right with a rate of associated anomalies of **20%** (chromosomal Abnormalities, neural tube defects & congenital heart Disease)

- The larger the hernia & the earlier it occurs, the more severe pulmonary Hypoplasia



The lateral chest radiograph demonstrating an air-filled mass extending into the anterior mediastinum is pathognomonic



- 2- Radiopaque hemithorax (if the bowel does not contain a significant amount of gas or if the left lobe of the liver occupies the majority of the hemithorax)
- 3-loss of normal ipsilateral diaphragmatic contour
- 4- Bowel in the thorax
- 5-Contralateral mediastinal shift
- 6-A coiled nasogastric tube in the hemithorax

Treatment

Surgical after preoperative preparation

A-Preoperative

- 1- A NG tube → to decompress the stomach & prevent distention of the herniated bowel which would further compress the lungs.
- 2-Respiratory support (mechanical ventilator & never use oxygen mask) for hypoxemia, hypercapnia & acidosis (before & after repair)

B-Surgery

- 1-A subcostal abdominal incision (some prefer thoracic approaches especially for right-sided defects either open or thoracoscopically)
- 2- The herniated bowel reduced from the pleural space.
3. Close the defect by nonabsorbable sutures.
 - A synthetic material is required to close large defects.
- 4- Placement of a chest tube in the pleural space (optional), if used → it is connected to a water seal and not to vacuum

C-post operative

Mechanical ventilation

Repair of the diaphragmatic defect is NOT a surgical emergency & should be done once the infant is stable & has minimal or no pulmonary hypertension

In the asymptomatic patient: (no pulmonary hypoplasia or hypertension)

- Repair is needed due to the risk of bowel obstruction.
- The viscera are reduced and any associated hernia sac excised.
- The defect is closed by suturing the posterior rim of diaphragm to the posterior rectus sheath since there is no anterior diaphragm.
- Rarely, a prosthetic patch closure is required

EVENTRATION OF THE DIAPHRAGM

Definition

Is an abnormally elevated &/or attenuated portion of the diaphragm

Clinical picture

The elevated hemidiaphragm → abnormalities of chest wall mechanics → impaired pulmonary function → Respiratory distress & pneumonia

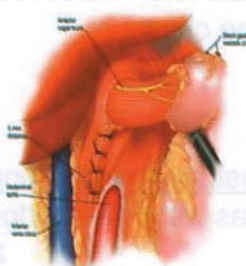
Investigations

- 1- Chest radiograph
- 2- Fluoroscopy or ultrasound demonstrate paradoxical movement of the diaphragm during spontaneous respiration.

Treatment

Surgical

- Indication: there are associated respiratory symptoms
- Method: plicating the diaphragm using interrupted nonabsorbable sutures



Etiology, either:

- 1- **Congenital** (usually idiopathic, but can be associated with congenital myopathies or intrauterine infections) → there is variable thinning or absence of diaphragmatic muscle
- 2- **Acquired** (as a result of phrenic nerve injury during forceps delivery or surgery).

ESOPHAGEAL ANOMALIES

Embryology

- The trachea and esophagus are derived from the primitive foregut.
- Initially, they appear as a common ventral diverticulum at about the 19th day of gestation → several days later, elongation & separation of the diverticulum into airway & esophagus (in a caudal to cephalic direction)
- Errors in this → in esophageal atresia, tracheoesophageal fistula & their variants.

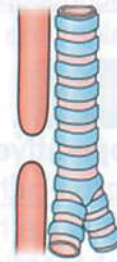
Classification

A- With Esophageal Atresia

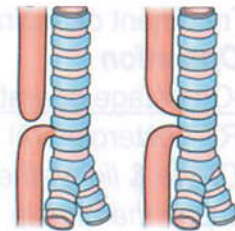
- 1-There is a blind proximal pouch & a fistula between the distal end of the esophagus & the distal 1/3 of the trachea (**type C, 85%**)
- 2- There is a blind proximal esophageal pouch, no tracheoesophageal fistula & a blind, short distal esophagus (**Type A, 10%**) → called pure or long gap atresia
- 3- There are fistulas between both proximal & distal esophageal segments and the trachea (**Type D, 2%** of cases).
- 4-There is a fistula between the proximal esophagus & the trachea and a blind distal esophagus without fistula (**Type B, 1 %** of cases).

B- Without Esophageal Atresia

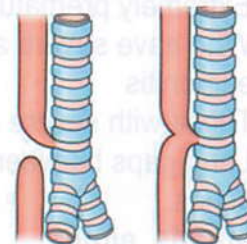
- 1- There is an H type tracheoesophageal fistula that is usually present in the low cervical region (**Type E, 4-5%** of cases).
- 2- There is esophageal stenosis consisting of a membranous occlusion (often containing cartilage) between the mid & distal third of the esophagus (rare).
- 3- There is a laryngo-tracheo-esophageal cleft of varying length, consisting of a linear communication between these structures (very rare).



Gross	A
Vogt	II



C	D
IIIb	IIIa



B	E
III	H-type

Clinical picture

- 1-Shortly after birth → pseudo-excessive salivation
 - 2- Repeated episodes of coughing, choking & cyanosis.
 - 3-Attempts at feeding → choking , gagging & regurgitation.
 - 4- Pneumonia
- (Infants with tracheoesophageal fistula in addition to esophageal atresia → reflux of gastric secretions into the tracheobronchial tree)

Investigations

1-Catheter test:

A size **10F** catheter passed into the esophagus through nose or mouth; if esophageal atresia is present → the tube will coil in the upper esophageal pouch & not go down the expected distance



2- Chest x-ray:

- If air present in the stomach & bowel → a tracheoesophageal fistula connects to the lower esophageal segment
- Absence of air below the diaphragm → no distal fistula
- Pulmonary infiltrates are usually noted first in the right upper lobe

3- Bronchoscopy

Determines the presence & position of the fistula

Treatment

A-Preoperative

1-A suction catheter should be placed in the upper esophageal pouch and the head of the bed elevated.

2-An echocardiogram

-to determine the position of the aortic arch since a right sided arch (present in 5%) → makes the standard right thoracotomy (or thoroscopic) repair difficult

3-Treatment of aspiration pneumonia (If possible)

B-Operation

1 -One stage operation

a) RT posterolateral thoracotomy with an extrapleural dissection.

b) Divide & ligate the fistula

c) Repair the atresia

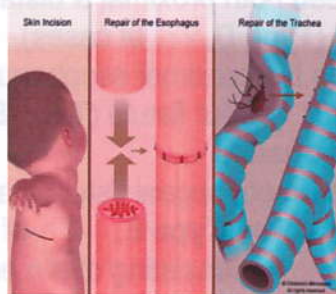
2-Staged operations

-Indication:

- Extremely premature babies
- Who have severe aspiration pneumonitis
- Those with severe anomalies
- Long gaps between the esophageal pouches.

-Methods, either:

- Cervical esophagostomy, division of the fistula & insertion of a gastrostomy tube → after several months → staged reconstruction by esophageal replacement with colon or stomach interposition
- A gastrostomy tube alone with intermittent bougienage & stretching of the upper esophageal pouch followed by → immediate interposition grafting & delayed primary esophageal anastomosis



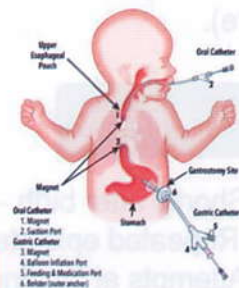
1- Associated anomalies (incidence **50%**):

- Cardiac (PDA, septal defects)
- GIT (imperforate anus, duodenal atresia)
- Genitourinary
- Skeletal

2-The **VACTERL** association (in **25%** of cases)

- Vertebral
- Anorectal
- Cardiac
- Tracheoesophageal
- Renal
- Limb

-Isolated esophageal atresia has been associated with various genetic abnormalities, including trisomy 18 & trisomy 21.
-Associated congenital anomalies should be treated



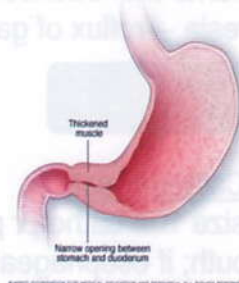
Hypertrophic Pyloric Stenosis (CHPS)

Incidence

- The male: female incidence is **4:1**
- The most common surgical disorder producing emesis in infancy.
- The disorder is more common in firstborn infants.
- Symptoms show seasonal variation → peak in spring & fall

Etiology

The cause is not known



Pathogenesis

Hypertrophy of circular & longitudinal muscularis of the pylorus and distal stomach antrum → progressive narrowing of the pyloric canal

Clinical picture

Symptoms

-Appear **2-4 weeks** after birth

1- Vomiting:

- Occasional regurgitation of some of the feedings → several days later → becomes more frequent and forceful.

- **No bile** and contains the previous feeding

- Shortly after vomiting, the infant acts starved & will feed again.

2-Constipation: The stools become infrequent & firm in consistency

3-Failure to thrive: progressive feeding intolerance → weight loss

Signs

General:

Dehydration → sunken fontanelles, dry mucous membranes & poor skin turgor

Local:

Inspection → Gastric peristaltic wave moving from the left costal margin to the area of the pylorus

Palpation →

Pyloric tumor or olive when the infant is relaxed (in over **90%**)

Complications

1- Dehydration

2- Hypokalemic hypochloremic alkalosis

Due to repeated vomiting with inadequate intake of formula

3- Starvation.

4- Gastritis & reflux esophagitis

5- Aspiration pneumonia

Investigations

1- **Abdominal ultrasound** (the most sensitive & specific test)

-Identify hypertrophic pyloric stenosis when:

a) The muscle thickness is **> 4 mm**

b) The length of the pylorus is **> 16 mm.**

2- **A contrast upper gastrointestinal series** (barium meal)

-indications: a) an experienced ultrasonographer is unavailable

b) The patient's symptoms may be not due to pyloric stenosis (eg, a premature, 1-week-old baby)

-A positive diagnostic signs are:

1. **String sign** → narrow pyloric channel

2. **A pyloric beak** → bulge in the distal antrum with streak of barium pointing towards the pyloric canal

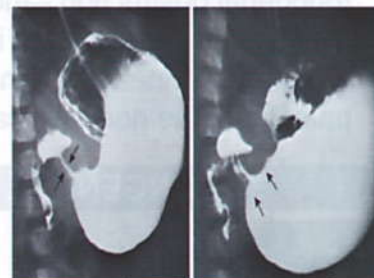
3. The **shoulder sign** (the pyloric mass bulges into the antrum)

4. **Complete obstruction of the pylorus**

The affected infant is full-term when born, feeds & grows well until 2-4 weeks after birth



An imaging study is indicated when the pyloric tumor cannot be palpated



INFANTILE HYPERTROPHIC PYLORIC STENOSIS.



Beak sign

Differential diagnosis

Repeated nonbilious vomiting in early infancy may be due:

- 1- Overfeeding
- 2- Intracranial lesions
- 3- Pylorospasm
- 4- Antral web
- 5- Gastroesophageal reflux
- 6- Pyloric duplication
- 7- Duodenal stenosis
- 8- Adrenal insufficiency

Treatment

- Operative by **pyloromyotomy**

Preoperative preparation:

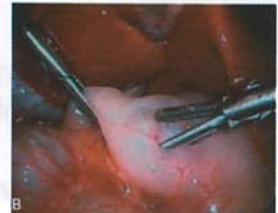
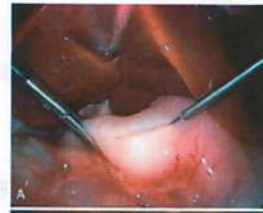
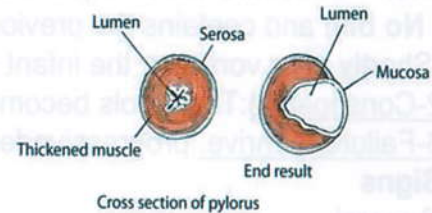
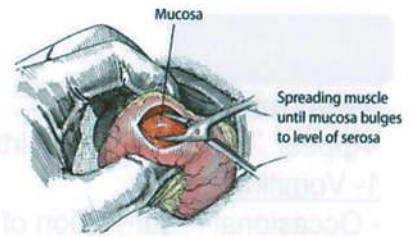
- 1- Correct dehydration
- 2- Correct hypokalemic hypochloremic alkalosis
- 3- Give normal serum chloride
- 4- Urine output > 1 cc/kg/h.

Operation

- 1- Pylorus is incised along its entire length
- 2- Spread widely exposing but not breaching the underlying mucosa

Approaches to pyloromyotomy: 3

- 1- RT. upper quadrant transverse skin incision (**Fredet-Ramstedt pyloromyotomy**)
- 2- Circumbilical skin incision
- 3- Laparoscopic approach.



Intestinal Obstruction In The Newborn

-Can be diagnosed prenatal by: **ultra sound** → polyhydramnios (Since fetuses continually swallow amniotic fluid into their GIT & excrete it in their urine)

-After birth, vomiting is the principal symptom

-Level:

a) If bile-stained → obstruction is distal to the ampulla of Vater.

b) Presence & degree of distention by physical examination (duodenal obstruction → no significant distention while colonic obstruction → massive as in Hirschsprung's disease).

-A careful perineal examination → determine the anus is present, patent & in the normal location

Duodenal atresia is diagnosed early in gestation rather than distal GIT atresia (due to mesenteric vascular abnormality) late in gestation.

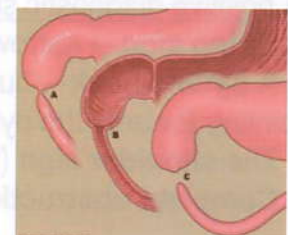
CONGENITAL DUODENAL OBSTRUCTION

Incidence

Duodenum atresia is twice as common as in the jejunum or ileum

Causes

- 1- Atresia (failure of recanalization)
- 2- Annular pancreas
- 3- Mucosal web (complete or variably perforate)
- 4- Stenosis
- 5- Preduodenal portal vein
- 6- Peritoneal bands (**Ladd's bands**) from malrotation.



Clinical picture

- 1- **Bilious emesis** (In **75%** of cases)
 - occurs shortly after birth and during attempted feedings.
- 2- Upper abdominal distension (rare)

Investigations

- 1- Plain abdominal x-ray
 - show air distended stomach and duodenum (**double bubble sign**)
 - Gas in the small & large intestine indicates incomplete obstruction.
- 2- Barium meal
 - Identify the presence or absence of malrotation in those cases with incomplete obstruction

Treatment

Surgery

- 1- RT upper transverse abdominal incision or via laparoscopy.
- 2- A Kocher maneuver should be performed with complete mobilization of 3rd & 4th portions of the duodenum.
- 3- Obstruction from Ladd's bands → simple bands division & correction of the malrotation.
- 4- Duodenal atresia & annular pancreas → Duodenoduodenostomy
- 5- A mucosal web → excised (avoid injury to the adjacent ampulla)
- In all cases → irrigate the distal bowel & assess for associated intrinsic obstruction and atresia (**1-3%** incidence).



- Obstruction from intestinal malrotation is a surgical emergency
 -Gastrojejunostomy should not be done because the blind duodenal pouch may cause repeated vomiting

Atresia & stenosis of the jejunum , ileum & colon

Etiology & Pathogenesis

In utero hernia, volvulus or intussusception → mesenteric vascular accident → aseptic necrosis and resorption of the necrotic bowel

Types

-Type I

A short area of necrosis → only stenosis or a membranous web occluding the lumen

-Type II

Extensive infarct → a fibrous cord between the two bowel loops

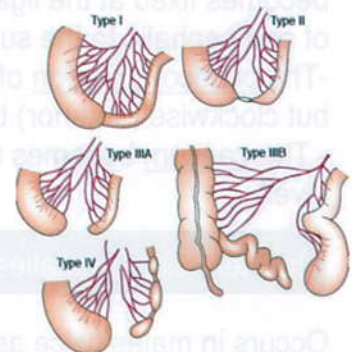
-Type III a

The proximal & distal bowel completely separated with a V-shaped defect in the mesentery

-Type III b (called **apple-peel** or **Christmas tree atresia**) in which There is a blind ending proximal jejunum, absence of a long length of mid small bowel & a terminal ileum coiled around its weak blood supply from an ileocolic vessel

-Type IV (in 10% of cases)

Multiple atresias occur



Clinical picture

- 1- Vomiting of bile
- 2- Abdominal distention
- 3- Failure to pass meconium

Investigations

1- The plain abdominal x-ray

Shows how far the obstruction exists along the intestine.

2- Barium enema

May be indicated to detect the level of obstruction

Treatment

Surgical

-Goals: 3

- (1) Restore the continuity of the bowel
- (2) Preserve as much intestinal length as possible
- (3) Retain the ileocecal valve if possible

The minimum length of bowel needed to sustain full enteral nutrition doubles in the absence of the ileocecal valve

Disorders Of Intestinal Rotation

Embryology

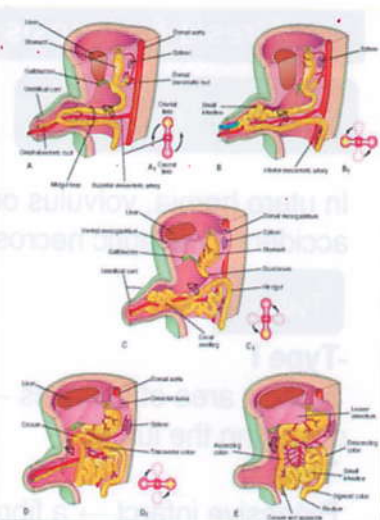
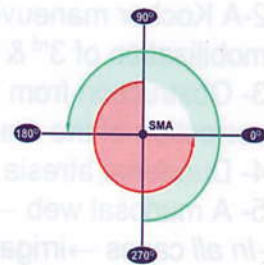
-The fetal intestine begins as a straight tube that grows faster than the abdominal cavity → thus herniates out into the body stalk (future umbilicus) at about **4th-6th weeks** gestation.

- At **10th-12th weeks** → the bowel returns to the abdominal cavity, rotates & becomes fixed to the retroperitoneum along diagonal axis extending from the level of the LT of the T12 vertebra to the level of the RT of the L5 vertebra.

-The duodenojejunal portion of gut rotates counterclockwise (posterior) to the superior mesenteric vessels for 270° & becomes fixed at the ligament of Treitz and located to the left of and cephalic to the superior mesenteric artery.

-The cecocolic portion of the midgut also rotates 270 degrees but clockwise (anterior) to the superior mesenteric artery.

- The caecum becomes fixed in the right lower abdomen (L5 level).



Incidence of anomalies of rotation & fixation

Occurs in males twice as common as in females

Classification

A-Nonrotation

- The midgut is suspended from the superior mesenteric vessels → small bowel is located on RT side & large bowel in LT abdomen.
- No fixation occurs & adhesive bands are not present.
- This is the fetal anatomy prior to 10 weeks' gestation.

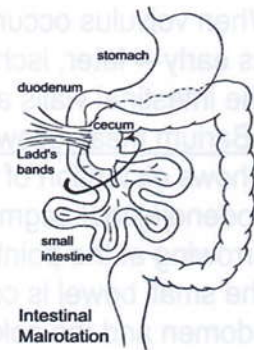
Nonrotation



- Because its base is so short → the mesentery is narrow → volvulus (with clockwise twisting of the bowel around the superior mesenteric)
- Usually found in patients with omphalocele, gastroschisis & congenital diaphragmatic hernia

B. Incomplete Rotation (called malrotation)

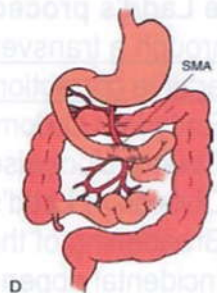
- May affect the duodenojejunal, the cecocolic segment or both.
- Adhesive bands (**Ladd's bands**) are usually present.
- In the most common form, the caecum stopped rotating and fixed near the origin of the superior mesenteric vessels → dense peritoneal bands extend from RT flank to the caecum → obstruct the 2nd or 3rd portion of the duodenum or other small bowel segments.
- The duodenojejunal segment also only partially rotates, usually stopping at or to the right of the vertebral bodies.
- The intestinal mesentery is fixed posteriorly but is very narrow (extends only between caecum & duodenojejunum) → volvulus



REVERSE ROTATION

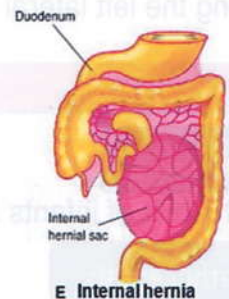
C. Reversed Rotation

- The bowel rotates varying degrees in a clockwise direction about the superior mesenteric axis.
- The duodenojejunal loop is anterior to the superior mesenteric A.
- The cecocolic loop may be prearterial or may be rotated clockwise or counterclockwise in a retroarterial position.
- The most frequent anomaly is retroarterial clockwise rotation → causes obstruction of the right colon.



D. Anomalous Fixation of Mesentery

- Leads to internal mesenteric & paraduodenal hernias, a mobile caecum or obstructing adhesive bands in the absence of anomalous bowel rotation.
- Excessive rotation of the duodenojejunal junction → makes the superior mesenteric artery compresses 3rd portion of the duodenum.



Clinical picture

Type of patient:

- The majority who develop intestinal obstruction are infants.
- Older patients may develop intermittent obstruction.

Symptoms

- Symptoms related to I.O (pain, vomiting, distension, absolute constipation)
- Peptic ulceration or malabsorption.
- In duodenal obstruction by bands → minimal abdominal distention
- Midgut volvulus → marked abdominal distention.

Signs

- Bloody stools & signs of peritonitis → indicates intestinal infarction

Investigations

1-Plain abdominal x-ray:

- With obstructing Ladd's bands → double bubble sign (mimics duodenal stenosis)

- Vomiting of bile occurs initially
- Adhesive bands → obstruction in the duodenum
- Volvulus → adhesions in upper jejunum

- Normal distribution but minimal amount of gas throughout intestine
- When volvulus occur → the proximal bowel will be distended with gas early → later, ischemic bowel resorbes gas → gasless abdomen
- The intestinal walls are thickened.

2- Barium meal follow through

- Shows distention of the duodenum, abnormal positioning of the duodenojejunal segment (usually to the right of the midline) & narrowing at the point of obstruction
- The small bowel is commonly visualized on the right side of the abdomen and the colon on the left.

3-Contrast enema: demonstrates abnormal position of the caecum.

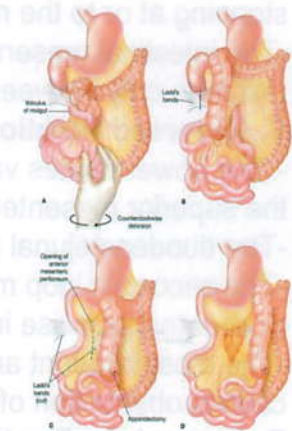


Treatment

The Ladd's procedure

-Through a transverse upper abdominal incision → **6** key elements in operative correction of malrotation:

1. Entry into abdominal cavity and evisceration
2. Counterclockwise detorsion of the bowel
3. Division of Ladd's caecal bands
4. Broadening of the small intestine mesentery
5. Incidental appendectomy
6. Placement of small bowel along the right lateral gutter & colon along the left lateral gutter



Meconium Ileus

Incidence

In 10-20% of infants born with cystic fibrosis

Pathogenesis

The thick mucus secretions of the small bowel → condense meconium → obstruction

Site: usually occurs in the terminal ileum.

Clinical picture

- 1- Normal birth weight
- 2- Very distended abdomen.
- 3-No meconium is passed
- 4- bilious emesis (occurs early)
- 5-Loops of thick, distended bowel may be seen & palpated

Complications

- 1- Repeated pulmonary infection with chronic bronchopneumonia, bronchiectasis, atelectasis & lung abscess.
- 2- Pancreatic insufficiency → malabsorption (requires pancreatic enzyme replacement)

Investigations

1-Plain abdominal X-ray:

- Show loops of bowel that vary greatly in diameter

- Meconium obstruction with no apparent cause has also been described in newborn infants

- Common complications of meconium ileus are related to the almost universal presence of cystic fibrosis



- The thick meconium → gives a ground-glass appearance.
- Air mixed with the meconium → the soap bubble sign (usually in the RT lower quadrant)

2-Contrast enema:

- will show microcolon with rare meconium flecks.
- Reflux of contrast through the ileocecal valve → shows small terminal ileum containing pellets of inspissated mucus, more proximally → meconium is packed → progressive distension.



Treatment

A-Non operative treatment (successful in 60-70% of cases)

- 1-A NG tube should be inserted and connected to suction.
- 2-A contrast enema (both diagnostic and therapeutic)
 - performed with a slightly hypertonic water-soluble contrast agent (never barium).
- 3-The addition of Nacetylcysteine (is mucolytic → disperse the meconium in uncomplicated cases)
- 4-Well hydration by IV fluids (during & after the procedure) → prevent hypovolemia caused by the hypertonic contrast solution

B-operative treatment → laparotomy

- Indication : failure of non operative treatment
- Steps: 1- The ileum is opened & if possible, flushed clear.
- 2- Bowel reanastomosis or bring it out as a double-barrel stoma. (Alternatively, a T-tube may be placed in the bowel & brought out of the anterior abdominal wall for postoperative irrigations).
- 3-Compromised intestine is resected
- 4- Appendectomy is performed (because of the high rate of appendicitis in patients with cystic fibrosis)



Hirschsprung's Disease

Etiology

- Is due to failure in the cephalocaudal migration of the parasympathetic myenteric nerve cells into the distal bowel.
- A familial association occurs in 5-10% of cases (more frequently when females are affected)
- Down's syndrome occurs in 10-15% of patients.

Incidence

- Males are affected 4 times more frequently than females (when the disease is limited to the rectosigmoid)

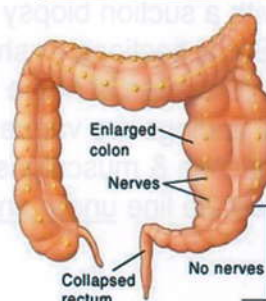
Site

- the absence of ganglion cells always begins at the anus & extends a varying distance proximally.
- Short-segment aganglionosis involving only the terminal rectum occurs in about 10% of cases
- The disease extends to the sigmoid colon in 75%
- More proximal colon in 10%
- The entire colon with small bowel involvement in 5%

-The length of involvement tends to be consistent in familial cases.

- Females tend to have longer aganglionic segments

- Extensive involvement of the small bowel is rare



Pathogenesis

The aganglionic bowel fails to relax in response to distention → functional bowel obstruction

Clinical picture

Symptoms

- 1-The infant passes little or no meconium within 24 hours.
- 2-Chronic or intermittent constipation
- 3-Progressive abdominal distention
- 4- Bilious emesis
- 5-Reluctance to feed
- 6- Diarrhea
- 7-Laziness
- 8-Irritability
- 9- Poor growth & development

-In older children,

- 1-Chronic constipation & abdominal distention are characteristic.
- 2-Passage of flatus & stool requires great effort
- 3- The stools are small in caliber

Signs

- A rectal examination →expulsion of stool & flatus with remarkable decompression of abdominal distention.

Investigations

- 1-Plain abdominal x-ray: show dilated loops of bowel
(Difficult to distinguish small & large bowel in infancy)

- 2- A contrast enema

- Do not clean out the stool before the fluoroscopic examination → will obscure the change in caliber between aganglionic & ganglionic bowel.

- Shows a narrow contracted (aganglionic) segment with dilated proximal bowel (have circumferential, smooth, parallel contractions (similar to those of jejunum) that are exaggerated contraction waves)
- Irregular, bizarre contractions (**saw-toothed pattern**) that do not encircle the aganglionic portion of the bowel

Radiographs of the abdomen & lateral pelvis should be repeated after 24-48 hours.

- The contrast agent will be retained for prolonged periods & saline enemas may be required to evacuate it.

- The delayed film may show the transition zone and the bizarre irregular contractions more clearly than the initial study.

- 3-Instrumental

- 1- **Rectal biopsy (most diagnostic).**

- Mucosal & submucosal biopsies taken from posterior rectal wall with a suction biopsy capsule without anesthesia at the bedside.

- Serial sections → show characteristic lack of ganglion cells & proliferation of nerve trunks in Meissner's plexus.

- If findings are vague → remove a **1 or 2 cm** full-thickness strip of mucosa & muscularis from the posterior rectum proximal to the dentate line under anesthesia → is sufficient for the pathologist to

Normally, the neonatal rectum is wider than the rest of the colon (including caecum), when the rectum is seen to be narrower than the proximal colon → Hirschsprung's disease suspected

- The symptoms vary widely in severity but almost always occur shortly after birth



-Hirschsprung's disease in patients who develop enterocolitis & diarrhea may mimic other causes of diarrhea

- Short segment Hirschsprung's disease may be difficult to differentiate from functional causes and rectal biopsy may be necessary

determine the presence or absence of ganglion cells in Meissner's plexus or in Auerbach's plexus.

2-Manometric studies (rarely performed except in older children)

-show a failure of relaxation of the internal sphincter following rectal distention by a balloon

Differential diagnosis

- 1- Rectal or colonic atresia
- 2-Meconium plug syndrome
- 3- Meconium ileus
- 4-Functional causes (as hypermagnesemia, hypocalcemia, hypokalemia & hypothyroidism), occur early in infancy & shows→
 - a) Stools normal in caliber
 - b) Soiling is frequent
 - c) Enterocolitis is rare
 - d) Stool is palpable in the lower rectum
 - e) Contrast enema shows uniformly dilated bowel to level of anus.

Treatment

A-Traditionally

- The surgical treatment was staged: a leveling colostomy→ after several months → resection of the aganglionic bowel & performance of a pull-through procedure.

B- Recently →a single-stage procedure (no colostomy) in newborn

This model is as follows:

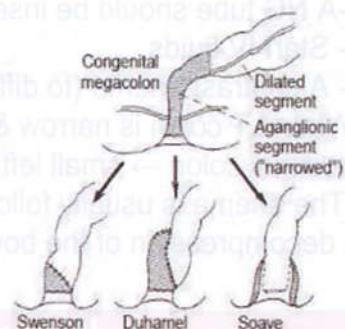
1- Bowel obstruction & enterocolitis (if present) → placement of a large (30F) rectal tube & repeated warmed saline irrigations in 10 mL/kg aliquots preoperatively.

2- Infants with moderate to severe enterocolitis → diverting colostomy, the correct (ganglionic) level for the stoma is established at time of surgery by frozen section analysis of the colonic muscle

3- Infants who are not ill → undergo anyone of **3** effective operations

- i) Swenson operation
- ii) Duhamel operation
- iii) Soave operation.

- Main principles: i) Removal of most or all of the aganglionic bowel
 ii) Preserving the surrounding nerves to the pelvic organs
 iii) Anastomosing ganglionic bowel (confirmed by frozen section analysis) to the rectum 0.5 cm above the dentate line.



-In contrast to the Swenson & Soave procedures, the Duhamel operation→ leaves a cuff of aganglionic rectum along which the ganglionic bowel is stapled→ creating a mini-reservoir.
 -A transanal mucosectomy has been used for those babies with short-segment disease.

NEONATAL SMALL LEFT COLON SYNDROME (Meconium Plug Syndrome)

Incidence

Two-thirds are male.

Pathology

This problem of newborn infants consists of low I.O. associated with:

- 1- left colon of narrow caliber
- 2- Dilated transverse and right colon.

Hypermagnesemia has been occasionally associated when the mother has been treated by IV magnesium sulfate for eclampsia

Clinical picture

Symptoms

- 1-Little or no meconium is passed
- 2- Progressive abdominal distention followed by vomiting.

Signs

- Rectal examination → normal or may reveal a tight anal canal.
- After thermometer or rectal stimulation by finger → some meconium & gas may be evacuated.

Investigations

Contrast enema:

- shows a very small left colon (to the level of the splenic flexure)
- Proximal to this point → colon & S.I. are greatly distended.
- In about 30%, a meconium plug is present at the junction of the narrow and dilated portion of the bowel and the enema (using water-soluble contrast) will dislodge it.

Differential diagnosis

- 1-Hirschsprung's disease
- 2- Meconium ileus.

These lesions rarely cause obstruction at the level of the splenic flexure

Treatment

- 1-A NG tube should be inserted
 - 2- Start IV fluids
 - 3- A contrast enema (to differentiate the various causes of low I.O.)
- When LT colon is narrow & contrast refluxes into the dilated proximal colon → small left colon syndrome.
 - The enema is usually followed by evacuation of copious meconium & decompression of the bowel.



-After enema, if colon:
a) Decompresses without further obstruction → Hirschsprung's disease is unlikely
b) Incompletely evacuated of meconium or symptoms persist → take a suction rectal biopsy to rule out Hirschsprung's disease

Imperforate Anus

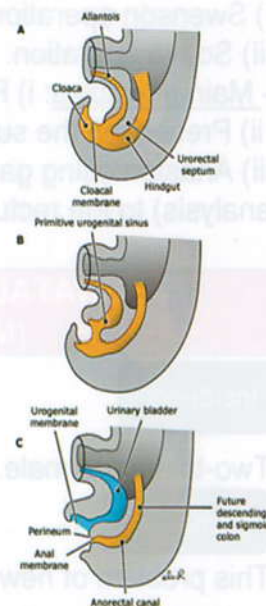
-The normal continence mechanism for bowel control consists of:

- 1- **Smooth muscle** → internal sphincter
 - 2- **Striated muscle** → complex of levator ani & external sphincter.
- The striated muscles assume a funnel shape, originating from the pubis, pelvic rim & sacrum → converge at the perineum while fusing with the internal and external sphincters.
 - Most of the striated muscle complex consists of:
 - a) Horizontal muscles → contract against the wall of rectum & anus
 - b) Longitudinal muscle fibers (cephalocaudal) → elevate the anus.

Embryology

1-Anomalies of the anus:

- result from: abnormal growth & fusion of embryonic anal hillocks.
- The rectum is normally developed & the sphincters usually intact.
- With proper surgical treatment, the sphincter will function normally.



2-Anomalies of the rectum

- result from: faulty division of the cloaca into the urogenital sinus and rectum by the urorectal septum.
- The internal sphincter & striated muscle complex are hypoplastic → surgical repair results in varying degrees of continence.

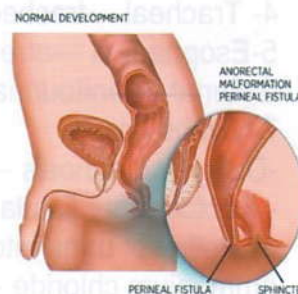
Classification

A-Low Anomalies

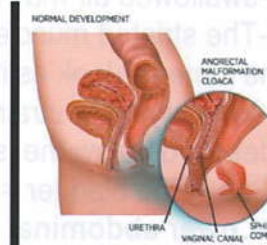
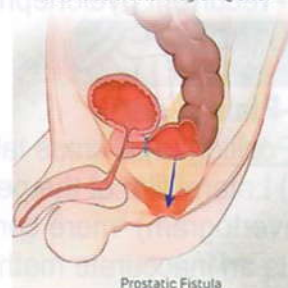
- Site of anus: anterior to its normal position or in normal position with a narrow outlet due to stenosis or an anal membrane.
- There may be no opening in the perineum but the skin at the anal area is heaped up and may extend as a band in the perineal raphe completely covering the anal opening.
- A small fistula usually extends from the anus anteriorly to open in:
 - a) Raphe of the perineum, scrotum, or penis (in males)
 - b) Vulva (in females)
- Often have well-developed perineal & gluteal musculature
- Rarely have sacral vertebral anomalies

B. High Anomalies

- The rectum may end blindly (**10%**) but more commonly there is a fistula to the urethra or bladder in the male or the upper vagina in the female.
- Often have deficient pelvic & gluteal innervation and musculature → a poor prognosis for continence after surgical repair
- Have high incidence of sacral anomalies
- The most severe of the high deformities is a cloacal anomaly (there is a common channel between the poorly developed pelvic structures "urogenital sinus & rectum" with a single perineal opening)



Pediatric Colorectal Program @CHOP



Clinical picture

- By physical examination:

A) **In low anomalies** → an ectopic opening from the rectum can be detected in the perineal raphe (in males) or in the lower vagina, vestibule or fourchette (in females)

B) **In high anomaly** →

- 1-No orifice or fistula can be seen upon examination of the perineum
- 2-Meconium is found at the urethral meatus, in the urine, or in the upper vagina.
- 3- Absence of external sphincter contraction with cutaneous stimulation of the anus (differentiate between high and low lesions)

Complications

- Associated anomalies occur in up to **70%** with a high anomaly.

- The associated VACTERL syndrome includes:

- 1- **V**ertebral → hemivertebrae or agenesis of one or more sacral vertebrae (agenesis of 81, 82 or 83 → corresponding neurologic deficits → neuropathic bladder & greatly impaired continence)
- 2- **A**nal → imperforate anus

3-**C**ardiac → anomalies of the heart (VSD, ASD, fallot)

4- **T**racheal → tracheo esophageal fistula

5-**E**sophageal → atresia

6-**R**enal → genitourinary anomalies (up to 50%)

7- **L**imbs / digits.

-Delay in diagnosis → excessive large bowel distention & perforation.

- Rectourinary fistula →

a) Reflux of urine into the rectum & colon → absorption of ammonium chloride → acidosis.

b) Colon contents will reflux into the urethra, bladder & upper tracts → recurrent pyelonephritis

Investigations

1-Plain X-ray

-Position: a) A cross table lateral film or

b) Lateral film of the pelvis with the baby inverted (Wangensteen invertogram) "more commonly used"

- Is an inaccurate method of establishing the lower extent of the rectum because:

1-swallowed air may not have completely displaced the meconium

2-The striated muscle complex may be contracted → obliterating the lumen → look as if the gas in the rectum ends high in the pelvis.

3-With crying or straining → puborectalis muscle & rectum may descend below the ischium → falsely low estimate of rectal height.

-Gas in the bladder = a rectourinary fistula.

2-Lower abdominal & perineal U/S, CT& MRI

- Define pelvic anatomy & location in relation to rectal musculature.

3- Lumbosacral spine MRI

-identify spinal cord anomalies such as a tethered filum terminale

Treatment

-The main goals of treatment:

(1) Relieve obstruction & allow passage of stool

(2) Place the rectal pouch on the perineum in good position

(3) Close the fistula.

A. Low Anomalies

- Approach: perineal

- Timing: in the newborn period

- Method:

1-Use muscle stimulator to determine the site of sphincter complex. 2-The anteriorly placed anal opening is completely mobilized and transferred to the normal position.

-After healing → the anal opening dilated daily for **3-5 months** to prevent stricture formation and to allow for growth.

B. High Anomalies

I- In the past

-Treated by a **three-stage** repair consisting of:

1- Colostomy and mucous fistula formation

2- Posterior sagittal anorectoplasty (**after 4-6 weeks**)

3 -Closure of the colostomy (**after several months**)

Anomalies of vertebrae & UT occur in 2/3 of all patients with high anomalies and in 1/3 of males with low anomalies.
-Vertebral abnormalities in females indicate a high imperforate anus.



II-Recently

- A **one-stage** repair has been performed by both posterior sagittal and Laparoscopic approaches.
- Continence is most dependent upon a functioning striated muscle complex (because anal sphincters are poorly developed especially internal sphincter) → requires conscious voluntary contraction.
- Preserve the afferent & efferent nerves of the defecation reflex arc as well as the existing sphincter muscles.
- In all cases, the surgically created anus must be dilated for several months to prevent circumferential cicatrix formation.

Intussusception

Definition

Telescoping of a segment of bowel (intussusceptum) into the adjacent segment (intussusceptent)

Incidence

- The commonest cause of I.O. in children bet. 6 months & 2 years
- The ratio of males to females is **3:2**.
- The peak age is in infants **5-9 months** of age (**80%** of patients are **< 2** years old)

Types

1-ileocolic (the most common)

- intussusception of the terminal ileum into the right colon

2-Ileo-ileal 3- Ileocolic 4-Colo-colic

Etiology

1-**Idiopathic (In 95%)**

2- **Adenovirus infections**

- Evidence:

a) In most cases, hypertrophied Peyer's patches are noted on the leading edge of bowel

b) The disease is most common in midsummer & midwinter.

3-**Mechanical factors** such as Meckel's diverticulum, polyps, hemangioma, enteric duplication, intramural hematoma (Henoch-Schönlein purpura) & intestinal lymphoma

4-**Postoperative intussusception** (can occur at any age)

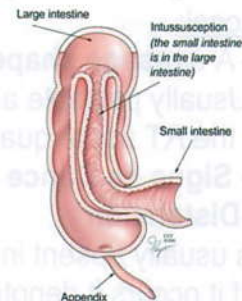
-Site: is usually ileoileal or jejunojejunal

-Cause: differential return of bowel motility (often after retroperitoneal surgery)

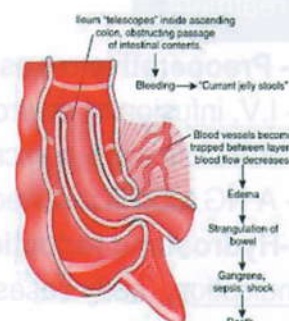
Clinical picture

Type of patient

Well nourished infants at the age of 3-12 months (age of weaning).



Any infant having colicky abdominal pains with the passage of blood-stained mucous per rectum → suspected of having intussusception



Symptoms

1-Abdominal pain

- awakens from sleep by severe abdominal colics, screams and draws his knees up onto the abdomen.
- The pain occurs in episodes that last for about 1 min. alternating with intervals of apparent well-being healthy child asking for food

2- Vomiting (in 85%)

- Early → Reflex vomiting from pain, but vomiting
- Late → due to bowel obstruction

3- Currant jelly stool.

- passes per rectum mucus & blood (from venous infarction)

Signs

- General: 1-Pallor and sweating (common during colic)

- 2- Progressive dehydration (from repeated vomiting & obstruction)

-Local:

1- A sausage shaped mass

- Usually palpable along the distribution of the colon most commonly in the RT upper quadrant of the abdomen.

2- **Signe de Dance** (emptiness in the right iliac fossa).

3-Distension

- is usually absent in early cases.
- If it occurs it denotes possible perforation or gangrene.

4-DRE

- Reveals bloody mucous in 60% of cases.
- Sometimes the head of the intussusception can be felt.

Investigations

1- Ultrasound examination.

2- Barium enema: (diagnostic and therapeutic in 60-80 %)

- Indication: In cases of doubtful diagnosis

- shows: **claw sign** (characteristic)

The barium will fill the colon & stops at the area of intussusception and a cylindrical filling defect appears with a trace of barium on either side

3-CBC → shows anaemia

Treatment

A- Preoperative resuscitation

- 1- I.V. infusion of dextrose and saline.
- 2- Antibiotics are prescribed.
- 3- A NG tube is inserted.

B-Hydrostatic reduction

- Indication: early cases

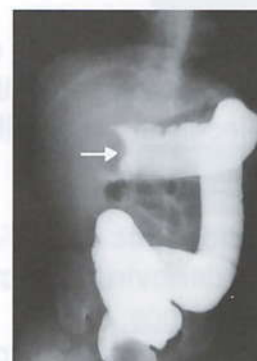
- Prerequisite: resuscitated patient

- Method: using contrast enema (either barium or air)



Complications:

- Prolonged intussusception → edema & hemorrhagic or ischemic infarction of intussusceptum → gangrene



-If **barium** is used → the column of contrast should not stand more than **100 cm** above the patient (to minimize the risk of perforation)

-**Air** is pumped into the colon at a pressure of **60-80 mm Hg**
(Never more than **120 mm Hg**)

- Success of reduction is confirmed by:

1-Free flow of barium into the small intestine for more than 5cm

2- Prompt clinical improvement with no further colics

-The baby should be kept under observation for **24 hours**.

- Success rate: is **75-95%**.

- Contraindications: 1- Doubtful diagnosis. 2- Late cases.

3- There is abdominal distension or rigidity (suspected peritonitis)

C- Surgery

-Indications: 1-hydrostatic reduction fails

2-Presence of perforation or peritonitis from the start

-Procedure: **squeeze method**

- At laparotomy, the head of the intussusception is squeezed backwards out of the containing colon.

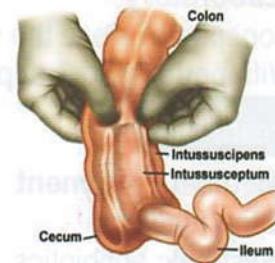
Prognosis

-Mortality is high in gangrenous cases.

- Recur in 2% of cases

-The proximal ileum should never be pulled backwards to avoid intestinal tears.

- If intussusception is irreducible or gangrenous → bowel resection & anastomosis.



Necrotizing Enterocolitis

Incidence

-Increasing due to the therapeutic advances in neonatal ICU → allow more premature smaller and smaller babies to survive.

- **80 %** occur in premature infants weighing < 2500 g at birth & **50%** are < 1500 g.

- May also occur in full-term infants.

Pathology

- Necrosis, ulceration & sloughing of intestinal mucosa → progresses to full-thickness necrosis and perforation.

-Gas-producing bacteria in the intestinal wall → pneumatosis

Pneumatosis may be noted on gross examination & on plain abdominal radiographs

Clinical picture

Symptoms

1-bilious vomiting

2-abdominal distention

3-bloody stools

Signs

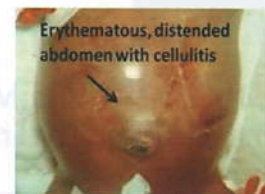
-General: lethargy and poor skin perfusion.

-Local:

1- Abdominal distention and fixed loops of intestine

2- Guarding (when intestinal perforation occurs but not obvious in weak premature infants)

3-Abdominal wall erythema, edema & crepitus (in case bowel necrosis is present)



Investigations

A-Radiological

1-Plain x-ray

- Position: Supine and cross-table lateral abdominal
- Show: a) early → small bowel distention
- b) Later → by pneumatosis intestinalis.
- c) Gas within the portal venous system can be seen but it is fleeting.
- d) Serial examinations may show a loop or loops of bowel that are fixed in position and dilated.

2- Paracentesis

- Indication: Infants who develop ascites without pneumoperitoneum
- Examine the fluid for bacteria (signify perforation)

B-Laboratory

- Nonspecific since the white blood cell count may be low or high
- With perforation & sepsis → thrombocytopenia & acidosis

Treatment

A-Medical Treatment

- 1- Stop feedings
- 2- Orogastric suction
- 3- Systemic antibiotics
- 4- Correct hypoxemia, hypovolemia, acidosis & electrolyte imbalance

B-Operation

-The only absolute indication is pneumoperitoneum.

-Procedure:

- 1-Laparotomy
- 2- Resect necrotic bowel
- 3-The proximal bowel is made into a stoma.

-Complications:

- 1-Severe disease → extensive bowel resection → short bowel syndrome.

C- Bedside drainage of the peritoneal cavity

-Indication: in very low birth weight infants <1500 g

; Is gaining acceptance for documented perforation

-Method: is in the right lower quadrant using local anesthesia

Prognosis

- 1/3 of cases → resolve without further treatment
- The overall survival rate is **>50%**.



- Perforation with peritoneal air develops in 20% of cases
- Contrast studies are hazardous & contraindicated as they may lead to Perforation
- Rarely primary anastomosis is safe
- Intestinal stricture may occur as a late complication following healing. So → contrast enema is used to evaluate the defunctionalized distal bowel before closing the stoma

Gastroschisis

Definition

Is a defect in the abdominal wall that usually occurs to the right of a normal insertion of the umbilical cord.

Incidence

Twice as common as omphalocele & the defect is usually smaller



Clinical picture

- 1-The small & large bowel, stomach, and often the fallopian tube/ovary/testis herniate through the abdominal wall defect.
- 2-Liver never present in the defect (unlike an omphalocele)
- 3- The bowel wall is edematous and has a very thick, shaggy membrane (peel) covering (as it is bathed in the amniotic fluid & compressing the mesenteric blood supply at the abdominal defect)
- 4-The loops of intestine are usually matted together & the intestine appears to be abnormally short.

Treatment

- 1-Urgent repair is necessary (Unlike omphalocele)
- 2-Small defects → closed primarily after manually stretching the abdominal cavity.
- 3-A staged approach is frequently required using a silo (described with Omphalocele).

-It is believed to arise at the site of involution of the RT umbilical vein

- Complications:

Since the bowel has not been contained intra-abdominally, the abdominal cavity fails to enlarge → cannot accommodate the protuberant bowel.

Liver & biliary tract disorders

Jaundice in the first 2 weeks of infancy is usually due to indirect (unconjugated) hyperbilirubinemia, The causes include:

1) Physiologic jaundice

Due to immaturity of hepatic function (associated with breast feeding)

2) Rh, ABO & rare blood group incompatibilities (produce hemolysis)

3) Infections.

-Jaundice that persists beyond the first 2 weeks in which the indirect and direct bilirubin levels are elevated → potential surgical disorders as:

1- Biliary atresia (The most frequent, **60%**)

2-Various forms of hepatitis (in **35%**)

3- Choledochal cyst is found (in **5%**)

-Mild indirect hyperbilirubinemia occurs with pyloric stenosis & disappears after pyloromyotomy.

-I.O. can intensify jaundice by increasing the enterohepatic circulation of bilirubin.

-Jaundice is an early & important sign of septicemia in the newborn

Biliary atresia

Definition

Is the absence of patent bile ducts draining the liver

Incidence

1 per 10.000 live births

Etiology

1-Is unknown but may due to inflammatory process → occlusion of variable lengths of biliary tree.

2- Congenital onset

-due to familial cases & frequent association with the polysplenia syndrome

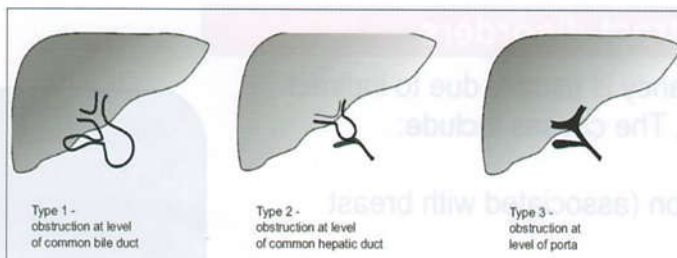
Pathology

- The atretic ducts consist of solid fibrous cords that may contain occasional islands of biliary epithelium.
- Liver biopsy → proliferation of the bile canaliculi containing inspissated bile → the failure to excrete bile out of the liver → progressive periportal fibrosis & obstruction of the intrahepatic portal veins → biliary cirrhosis .

Types

- **Type 1:** Obstruction at the level of common bile duct (CBD)
- **Type 2:** Obstruction at the level of common hepatic duct (CHD)
- **Type 3:** (unfortunately **90%** of cases) → **Non correctable** the extrahepatic portion of the biliary tract is occluded and appears as a cord like structure.

Correctable



Clinical picture

- 1- Jaundice is noted at 2-3 weeks of age.
- 2- Urine may be dark
- 3- Stools may be normal or clay-colored & may contain an increased quantity of fat but are of normal consistency and not frothy.
- 4- The liver: a) Early → may be of normal size
b) Later → becomes enlarged
- Progressive cirrhosis → hard liver
- 5- Splenomegaly usually develops.

Investigations

A-Laboratory

1-liver function tests

- The direct bilirubin levels >3 mg/dL.
- Alkaline phosphatase levels are often elevated to 500-1000 units/L
- Glutamyltranspeptidase levels are > 300 units

2-CBC

3- Metabolic & serologic screening.

B-Radiological

1-Ultrasonography

Show absence or inability to visualize a contracted gallbladder

-Biliary atresia probably develops after birth because:
a) jaundice is not remarkable in the newborn period but evident > 2 weeks later.
b) conjugated bilirubin is not cleared by the placenta (unlike unconjugated bilirubin) and jaundice due to conjugated hyperbilirubinemia with biliary obstruction has not been recognized in newborn infants



2-Radionuclide scanning (HIDA)

-Using technetium Tc 99m-labeled iminodiacetate compounds to observe the intensity of uptake within the liver and evidence of secretion into the bowel

-Usually preceded by a 2- to 3-day course of phenobarbital → promote tracer uptake.

3-Needle biopsy of the liver

- Performed at any age if the clotting tests are normal



Differential diagnosis

1-Choledochal cyst: - Identified by the presence of a palpable mass in the RT upper quadrant and ultrasonographic confirmation.

2- Inspissated bile syndrome , is due to:

a) Haemolytic process → a large bilirubin load is excreted in the bile ducts → becomes coalesced and impacted

b) After a prolonged period of bowel rest with TPN

-Recognized by abdominal ultrasound

3-Hepatitis: -Most commonly of unknown cause.

-May be due to a variety of infections of maternal origin such as toxoplasmosis, cytomegalovirus, rubella syndrome, herpes simplex, coxsackievirus & varicella

-Serum should be tested for elevated antibody titers to these agents

4- Genetic metabolic diseases.

-include antitrypsin deficiency, galactosemia & cystic fibrosis.

-Other rare causes include sepsis, parenteral alimentation cholestasis, Gilbert's disease & Alagille's syndrome

5- Neonatal physiologic jaundice

Is self-limited and also responds to phototherapy

Treatment

Surgical exploration

-Timing: as early as possible in infancy when biliary atresia is the likely cause.

- Delayed treatment → progressive cirrhosis.

- Cholangiography is performed by cannulation of the gallbladder (through a transverse abdominal incision or via laparoscopy) and gentle instillation of a water soluble contrast agent

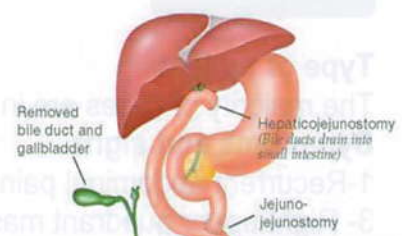
I- In the rare correctable type → hepaticojunctionostomy is performed.

II- Confirmed biliary atresia → requires hepatic portoenterostomy (**Kasai procedure**).

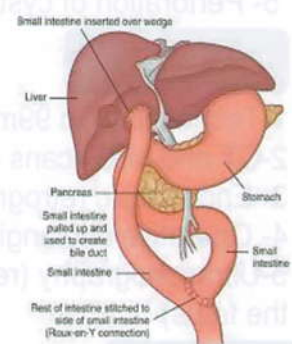
1- The scarred bile ducts and gallbladder are removed

2- A Roux-en-Y limb of jejunum is sutured to an area of the hilum bounded laterally by the hepatic artery branches.

-If this fails or the child develops significant complications of chronic liver disease → liver transplantation will be required.



Roux-en-Y Hepaticojunctionostomy Procedure performed for cholangiocarcinoma and biliary injuries.



Choledochal Cyst

Definition

It is dilation or diverticulum of all or a portion of the common bile duct.

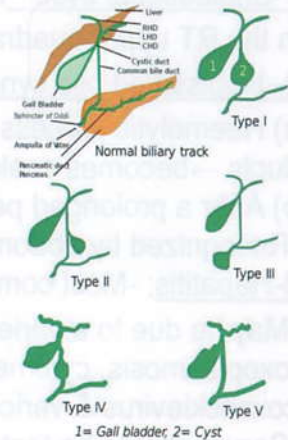
Etiology is unknown

Incidence

- There is a female predominance (3:1)
- More common in Asians with a large majority of the reported cases from Japan.

Types

- **Type I** is a fusiform dilation of the extrahepatic bile duct.
- **Type II** is a saccular outpouching of the common bile duct.
- **Type III** is referred to as a choledochoceles and is a wide-mouth dilation of the CBD at its confluence with the duodenum.
- **Type IV** is cystic dilation of both the intra- & extrahepatic bile ducts.
- **Type V** consists of lakes of multiple intrahepatic cysts with no extrahepatic component (when associated with hepatic fibrosis → termed **Caroli's disease**)



Clinical picture

Type of patient

The majority of cases are in childhood before 10 years of age

Symptoms and signs

- 1-Recurrent abdominal pain
- 2-Episodic jaundice
- 3- Right upper quadrant mass.
- 4- As children grow older, the cyst may become infected → painful & recurrent attacks of pyrexia
- 5- Perforation of cyst → bile peritonitis (rare)

If left untreated →
cause cholangitis &
cholangiocarcinoma

- The diagnosis is most often established by the clinical presentation & abnormal U/S

Investigations

- 1-Techetium Tc 99m-labeled HIDA scan
- 2-CT and MRI scans
- 3- Endoscopic retrograde cholangiopancreatography (ERCP)
- 4- Operative cholangiography.
- 5-Ultrasonography (responsible for detecting choledochal cysts in the fetus)

Treatment

The cyst should be excised and hepaticojejunostomy is performed

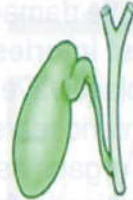
Anomalies of the gallbladder

- 1- Absence of the gall bladder (rare, in 0.03% of cases)
- 2- The phrygian cap (in 2 -6 %)
- is kinking of the fundus of the gall bladder
- It is of no clinical significance.
- 3- Double gall bladder (in 1 in 4000)
- with two separate cavities and cystic ducts
- 4- Floating gall bladder.
- Hangs on a mesentery → liable to undergo torsion.
- 5- Left sided gall bladder (extremely rare)
- 6- Intrahepatic gall bladder
- Is associated with an increased incidence of gall stones



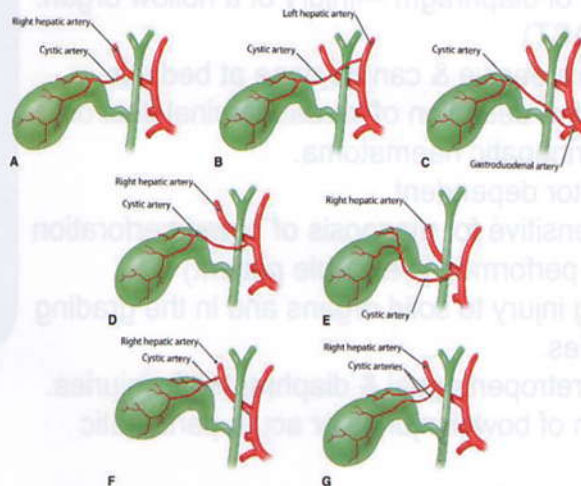
Cystic Duct Anomalies

- 1- Absence of the cystic duct
- Injury of the CBD is liable to occur when cholecystectomy is performed in this patient
- 2- Low insertion of cystic duct (common)
The cystic duct opens into the common duct near the ampulla.
- May lead to ligation of CBD during a cholecystectomy operation.
- 3- The cystic duct may enter the right hepatic duct
- RT hepatic duct may be mistaken for the cystic duct and ligated



Anomalies of the Hepatic and Cystic Arteries

- 1- Accessory cystic artery (**the most common**)
- May be torn if it is not identified.
- 2- A large accessory left hepatic artery (in about **5%** of people)
- May arise from the left gastric artery.
- 3- The right hepatic artery may arise from the SMA artery.
- 4- The RT hepatic artery pass in front of or behind the CHD or CBD
- May be mistaken for the cystic artery → ligated.



Review Subjects

Abdominal Injuries

Mechanism of injury

1. Blunt injuries

- cause: direct blows to the abdomen or RTA (more common)
- Common sites: the renal pedicle, the duodenojejunal flexure, the ileocaecal area or the neck of the pancreas.

2. Penetrating injuries

- Causes: stabs or gunshots.
- The severity of injury with gunshots is determined by the formula: **(Kinetic energy = MV^2/g)**
(M → mass of bullet, V → velocity & g → acceleration of gravity)
- When a bullet strikes soft tissues → shock waves spread out → extensive damage to the surrounding tissue far from 1st tract

3. Blast injuries

- The blast waves → shear waves → submucosal hemorrhages, mesenteric tears or perforation near the ileocaecal area.
- Solid organs as the liver may be severely lacerated.
- In addition to the blast wave, the produced missiles after the explosion → different blunt or penetrating abdominal injuries.

Investigations

A-Laboratory investigations

- 1- CBC & haematocrit value:
decreasing Hb & haematocrit denote bleeding.
- 2- A rising leucocytic count points to peritonitis.
- 3- High serum amylase level suggests pancreatic injury.

B-Radiological

- 1- Plain chest & abdominal X-rays (only in hemodynamically stable)
 - i) May reveal fracture of lower ribs or pelvis or the presence of FB
 - ii) Free air under RT cupula of diaphragm → injury of a hollow organ.
- 2- Abdominal ultrasound (FAST)
 - i) is non-invasive, quick, inexpensive & can be done at bed site.
 - ii) Sensitivity of **85-95%** for the detection of intraabdominal fluid or blood e.g. perisplenic or perihepatic haematoma.
 - iii) Disadvantages: a- Operator dependent
b- Not sensitive for diagnosis of bowel perforation
- 3- CT abdominal scan (only performed in a stable patient)
 - i) Very accurate in detecting injury to solid organs and in the grading and follow-up of these injuries.
 - ii) Sensitive in diagnosis of retroperitoneal & diaphragmatic injuries.
 - iii) Not sensitive in detection of bowel injuries or acute pancreatic injuries.

- In RTA → sudden deceleration → stress to areas of junction between freely mobile intraperitoneal organs & those with fixed retroperitoneal position
- Sudden compression of the abdomen → laceration of the liver or spleen.
- **Stab wounds to the abdomen**
 - a) 1/3 of cases non penetrating
 - b) 1/3 penetrating but not causing intraabdominal injury
 - c) 1/3 cause an intraabdominal injury
- Possible intra-abdominal injuries:
 - a) To solid organs e.g. liver, spleen or mesentery → internal Hge
 - b) To hollow organs e.g. stomach, duodenum, S.I or colon → peritonitis.
 - C) Retroperitoneal injuries e.g. pancreas, kidneys or major blood vessels.

C-Diagnostic peritoneal lavage (DPL)

Indications: Blunt abdominal trauma in an adult, associated with:

1. Suspicion of organ injury with equivocal signs.
2. Unreliable abdominal examination because the patient is unconscious, e.g., head trauma or drug / alcohol intoxication.
3. Unexplained hypotension that may be caused by blood loss.

Contraindications

1. Evident intra-abdominal organ injury that requires laparotomy.
2. Pregnancy.
3. Liver cirrhosis.
4. Severe obesity.
5. Prior abdominal surgery.

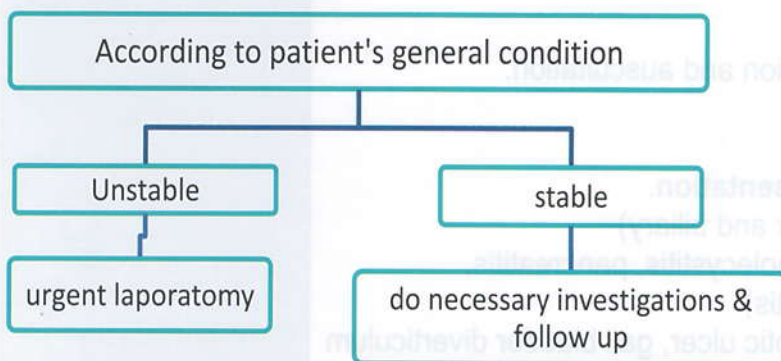
Procedure

1. Prepare abdomen with antiseptic solution then cover with sterile towels.
2. Local infiltration of local anesthetic, e.g. lidocaine in the midline below the umbilicus.
3. **2-3 cm** skin incision followed by a 1 cm incision in the linea alba.
4. Peritoneum is entered with a dialysis catheter.
5. The tube is directed posteriorly and inferiorly into the pelvis.
6. Aspiration with a syringe.
(Gross blood or gross enteric contents → immediate laparotomy)
7. If neither blood nor enteric content is aspirated, 1 L of warm saline is instilled into the peritoneum by intravenous tubing.
8. After waiting for 5 minutes the empty saline bottle is placed down in a dependent position to suckle out the lavage fluid
9. A sample of the fluid is sent to the laboratory.
10. Catheter is removed & linea alba & skin are closed with sutures.

D-Diagnostic laparoscopy (in hemodynamically stable patients)

Treatment

- 1-Follow the general measures for trauma patients (ATLAS)



N.B.: There is now a growing tendency to treat many patients with hepatic and splenic injuries conservatively

- Site of injury & localization of the physical signs give clue to diagnosis e.g. presence of hypovolaemic shock with rigidity & tenderness in RT hypochondrium denotes liver injury.
- Trauma to lower thoracic cage → splenic or hepatic injuries.
- In bowel injuries, C/P may not be apparent on initial examination.
- Positive findings in DPL that diagnose an intra-abdominal injury → require laparotomy are:
 - a. RBCs count $>10,000,000/ml$.
 - b. White blood cell count $>500/ml$.
 - c. Elevated amylase.
- Indications for Urgent laparotomy:
 1. General & local manifestations of intra-abdominal bleeding, e.g., pallor, tachycardia, hypotension with abdominal rigidity, tenderness & distension.
 2. General & local manifestations of peritonitis.
 3. Stab wounds with a protruding viscus (injury is deeper than parietal peritoneum)
 4. All missile injuries of the abdomen

Acute Abdomen

Definition

Acute abdomen is a term applied to acute abdominal pain.

Clinical picture

A-Personal history

The age, sex, marital status, residence and menstrual history

B-Symptoms

1. Acute pain (the most important)

-There are 2 types:

- a) Visceral pain
- b) Somatic pain
- May be referred to other regions:
 - a) renal or ureteric colic → to groin or testis, along → genitofemoral N
 - b) Biliary colic → to lower angle of RT scapula (7th-9th thoracic nerve)
- Sudden onset of pain → haemorrhage, perforation or torsion
- Gradual onset → inflammation.

2. Vomiting

-Onset: usually once or twice after the onset of pain.

- Frequent and profuse vomiting is serious.

- I.O. → Forcible vomiting (due to stomach & abdominal muscles contraction)

- Acute gastric dilatation, paralytic ileus or peritonitis → regurgitant vomiting (passive return of gastric contents without any force)

3. Bowels:

- a) Constipation (in most cases)
- b) Tenesmus (may be present in pelvic appendicitis)
- c) Bleeding per rectum (in Mesenteric thrombosis)

4. Urinary problems and menstrual disturbances

Signs

1. General: Vital signs (especially RR) and looking for pallor, jaundice & dehydration.

2. Abdominal examination:

by inspection, palpation, percussion and auscultation.

Causes

I-According to the mode of presentation.

1. Colics. (Intestinal, appendicular and biliary)
2. Inflammations (Appendicitis, cholecystitis, pancreatitis, diverticulitis & Meckel's diverticulitis)
3. Perforations (Of appendix, peptic ulcer, gall bladder diverticulum & typhoid ulcer of the small bowel)
4. I.O. (Simple, strangulation, and paralytic ileus)
5. Internal haemorrhage.
6. Urological causes (Calculi and inflammations)
7. Gynaecological causes
 - i) Mid menstrual pain.
 - ii) Twisted ovarian cyst.
 - iii) Dysmenorrhoea
 - iv) Ruptured ectopic pregnancy.
 - v) Ruptured ovarian cyst.
 - vi) Pelvic inflammatory disease.

- Visceral pain

Character: vague ill defined pain

Cause: distension of a hollow viscus. (felt in the segment of abdominal wall having the same nerve supply of the affected organ) , so

- Foregut pain (stomach & duodenum) → felt in the epigastrium.
- Mid-gut pain (jejunum to transverse colon) is felt in peri-umbilical region.

c. Hind-gut pain (transverse colon to anal canal) is felt in lower abdomen.

-Somatic pain

Cause: irritation of the parietal peritoneum → well localized over the affected organ.

-The presence of these 2 types of pain explains why pain in acute appendicitis :

initially → vague & in umbilical region,
later → localized to RT iliac fossa

- Do not forget to examine the spine, the scrotum, the hernial orifices and to do DRE

8. Medical causes of acute abdomen:

- i) Severe gastroduodenitis.
- ii) Mediterranean fever.
- iii) Activity of a peptic ulcer
- iv) Intermittent porphyria.
- v) Enterocolitis.
- vi) Basal pneumonia & pleurisy
- vii) Uraemia.
- viii) Acute MI
- ix) Diabetic ketoacidosis.
- x) Alcoholic hepatitis

II-According to site

A) Upper abdominal

- 1- Perforated PU
- 2- Leaking abdominal aortic aneurysm.
- 3- Biliary colic & acute cholecystitis.
- 4- Mesenteric vascular occlusion.
- 5- Acute pancreatitis.
- 6- Acute MI

B) Mid-abdominal

- 1- Mesenteric vascular occlusion
- 2- I.O.

C) Left lower abdomen

- 1- Colonic diverticulitis
- 2- Left ureteric colic
- 3- Colitis

D) RT lower abdomen

- 1- Acute appendicitis.
- 2- Mesenteric adenitis
- 3- Regional ileitis (Crohn's disease)
- 4- Right ureteric colic
- 5- Colitis
- 6- Meckel's diverticulitis

E) Pelvic

- 1- Mid menstrual pain
- 2- PID
- 3- Proctitis
- 4- Cystitis
- 5- Complicated ovarian cyst
- 6- Prostatitis
- 7- Ectopic pregnancy
- 8- Pelvic appendicitis

F) Abdominal and back pain

- 1- Biliary colic and acute cholecystitis
- 2- Acute pancreatitis
- 3- Renal and ureteric colic
- 4- Leaking abdominal aortic aneurysm
- 5- Posterior DU penetrating into pancreas.
- 6- Spine diseases with radicular pain that radiates forwards

Some common causes of acute abdomen

Acute cholecystitis

- Site & Character: initial pain is diffuse & colicky in the upper abdomen, Later → localizes in RT hypochondrium.
- It is difficult to palpate GB (20%) due to the overlying tenderness & rigidity.
- Ultrasound is diagnostic & shows :
 - 1- Gallstones in 95% of cases.
 - 2- Distension of the GB
 - 3- Thickened walls.
 - 4- Pericholecystic fluid collection.
 - 5- Localized tenderness

Acute diverticulitis

- Type of patient: rare < 40.
- Commonest site → Sigmoid
- Diagnosis relies mainly on C/P
- Investigations:
 - 1) Gastrografin enema
 - 2) Barium enema (postponed till after resolution of acute attack)
 - 3) CT → may reveal
 - i- Localized thickening of colonic wall
 - ii- Density in the pericolic fat
 - iii- pericolic abscess.

Acute appendicitis

- Type of patient: any age but is commonest in 2nd & 3rd decades.
- Site of pain: characteristic shifting from the centre to RT lower abdomen.
- C/P:
 - 1-Pain (always before vomiting)
 - 2-Diarrhoea is against the diagnosis.
 - 3- Anorexia & nausea (commonest)
- C/P may be variable (retrocaecal, pelvic, pregnancy and children).
- Investigations: (in difficult cases)
 - 1- **Leucocytic count**: Polymorph nuclear leucocytosis & shift to the left (If absent → not clue against appendicitis)
 - 2- **If a ureteric calculus is suspected** → do urine examination, plain X-ray UT, IVU & ultrasound
 - 3- **In females, if a tuba-ovarian or uterine problem is suspected** → do pelvic ultrasound and laparoscopy

Acute pancreatitis

- Site of pain: Severe epigastric pain that increases in intensity.
- Referral: to the back.
- C/P:
 - 1-Profuse vomiting (prominent) When the stomach becomes empty, the patient starts retching.
 - 2- The patient may be shocked.
 - 3- Slight tenderness & guarding
- Investigations:
 - 1- **Serum amylase**
 - rises within 12 hours & returns to normal within 2-3 days.
 - It has no predictive value.
 - Is raised in many other conditions
 - 2- Investigations:
 - 1- Plain X-ray of the abdomen
 - 2- CT → may reveal enlargement of the pancreas, peripancreatic fluid collection or pancreatic necrosis.

Perforated peptic ulcer

- Cause: May be related to the intake of NSAIDs or corticosteroids.
- The onset: is sudden,
- Perforated DU: GU = 6:1.
- The ulcer may be acute or chronic.
- Board like rigidity is present.
- C/P not marked in elderly
- Investigations:
 - 1- Plain X-ray of the chest in the erect position → free gas under the cupula of the diaphragm in **60- 85%** of cases.
 - 2-Gastrografen meal → leakage of the contrast.
- Differential diagnosis
 - 1- Causes of acute upper abdominal pain(mentioned earlier)
 - 2- Acute appendicitis.
- In some cases, perforated PU → leaking fluid → trickles along RT paracolic gutter to RT iliac fossa (simulate appendicitis)

Mesenteric Ischaemia

- This is type of strangulation I.O.
- Suspected in patients > 50 years with valvular or atherosclerotic heart disease, arrhythmias, hypotension, hypovolaemia, M.I., or polycythaemia.
- C/P:
 - 1-Haematemesis & melaena
 - 2-pain out of proportion to examination
- Investigations:
 - 1-Plain X-ray → ground glass appearance.
 - 2- U/S & CT scans → occluding thrombus, bowel wall edema or abnormal gas patterns.
 - 3- CT angiography.
 - In SMA thrombosis → occlusion of the orifice of the SMA
 - In SMA embolism → the embolus is lodged few cm.s from the orifice.
- Early thrombolysis by cannulation of SMA via transfemoral aortography.
- If C/P is suggestive of mesenteric ischemia & there are no facilities for these investigations → laparotomy

Perforated typhoid ulcer

- Perforation occurs during the 3rd or 4th week of fever.
- C/P: Fever, malaise & headache are present.
- Severe local pain & tenderness.
- Collapse is usually present.

Simple bowel obstruction

- C/P: 1-Colicky abdominal pains
2-Vomiting 3- Abdominal distension
4- Absolute constipation.
- Investigations:
1-Plain X-ray→ distended loops or fluid levels.
2- CT scan with oral contrast (in doubtful cases)
3-Barium enema (in large bowel obstruction)

Strangulation I.O.

- Severe pain which is persistent & not relieved by NG suction.
- Localized tenderness & guarding.
- Fever and tachycardia.
- Leucocytosis.

Ruptured ovarian cyst

- C/P: lower abdominal pain, tenderness & guarding.
- No toxæmia.
- Investigations:
Abdominal & pelvic U/S

Torsion of an ovarian cyst

- Severe lower lateral abdominal pain.
- Adnexal mass may be palpable.
- Ultrasound is diagnostic.
- Laparoscopy (diagnostic & therapeutic)

Pseudo-obstruction

- Includes a group of disorders which give C/P of I.O. but without a demonstrable organic lesion.
- Causes:
1- Elderly patients with CVS accidents.
2- Endocrine disorders, e.g., myxoedema.
3- Neurologic disorder, e.g., Parkinsonism.
4- Drugs, e.g., atropine like & tranquilizers.
5- Electrolyte disturbances, e.g., hypokalaemia.
6- Metabolic disturbances as in uremia & DKA
7- Shock, e.g., from burns & septicaemia.
8- Retroperitoneal irritation by collection of blood or urine, or by acute pancreatitis

Ruptured ectopic pregnancy

- Risk factors: prior salpingitis, tubal ligation, tubal repair, IUD and prior ectopic pregnancy.
- C/P:
1-History of menstrual abnormalities
2- Severe lower abdominal pain.
3- Pallor (usually a striking feature)
4-Abdominal examination reveals tenderness & guarding.
5- PV examination → tender cervix.
- Investigations:
1- Chorionic gonadotropin testing is positive.
2- Ultrasound.
Free blood is present in the pelvis.
(The presence of a gestational sac in the uterus is against the diagnosis)
3- Laparoscopy is very helpful

Pelvic inflammatory disease (PID)

- Suspected in females in the reproductive period.
- IUD increases the possibility.
- Inflammation is usually bilateral but may be severer on one side.
- Usual causative organisms: Streptococci, E. coli & anaerobes
- C/P:
 - 1- Lower abdominal tenderness & guarding with high pyrexia.
 - 2- leakage or rupture → Peritoneal signs in upper abdomen
 - 3- PV examination & movement of the cervix are tender.
- Investigations:
 - 1-High vaginal swab for culture & sensitivity.
 - 2- Ultrasound

Common acute abdominal conditions in infants and children

1- Acute appendicitis:

- a. The diagnosis is difficult & early perforation is common.
- b. Diarrhoea may be present.

2- Non-specific mesenteric lymphadenitis: These patients are usually diagnosed as acute appendicitis.

3- Intussusception: This presents by colicky abdominal pains, constipation & bleeding per rectum.

4- Intestinal volvulus.

5- Meckel's diverticulitis

6- Primary peritonitis.

Investigations of acute abdomen:

A-Laboratory investigations

1. CBC
2. Urea & electrolytes.
3. Blood sugar.
4. Serum amylase.
5. Urine analysis

B-Radiological investigations

1. Plain X-ray of the chest (in the erect position)

- In perforated viscus → free gas under the cupola of the diaphragm.
- A basal pneumonia will be detected.

2. Plain X-ray of the abdomen may reveal:

- a) Calculi of the urinary tract.
 - b) Distended loops of bowel or fluid levels in I.O.
3. Abdominal ultrasound can diagnose the following:
- A) Acute calcular cholecystitis(Stones are detected in **95%**). The GB is distended with thickened wall & subserosal edema
 - b) Acute pancreatitis (Enlargement of the pancreas, pancreatic pseudocyst or abscess & pancreatic necrosis)
 - c) Leaking aortic aneurysm.
 - d) Colic by ureteric stones →distended pelvicalyceal system
 - e) Gynaecological disorders (Ruptured ovarian cyst, twisted ovarian cyst, ectopic pregnancy & PID)

4. CT scan of the abdomen:

- The main advantage: its picture is neither affected by obesity nor by the presence of ileus.
- If performed with oral & IV contrast is very helpful for diagnosis of :

a) **Acute pancreatitis.**

Reveals extent pancreatic enlargement , presence of gas, fluid or blood around the pancreas, can detect pancreatic necrosis.

b) **Retroperitoneal haemorrhage.**

Modern investigations should not overwhelm good clinical judgment. -In the majority of cases accurate diagnosis is reached by clinical examination supplemented by few tests

c) Bowel infarction.

May show gas (intramural, in mesenteric veins or in portal vein)

-The bowel wall is thickened.

-The mesenteric thrombus may be visualized.

D) Splenic infarction.

E) Diverticulitis.

-Inflammation of the bowel wall with thickness is detected.

-An abscess can be visualized.

F) Localized fluid collection or free fluid in the peritoneal cavity.

G) Intestinal obstruction.

- It will reveal the site of transition between dilated & collapsed loops

-it may show the etiology of the obstruction.

C-Diagnostic laparoscopy:

is valuable especially for gynaecological problems.

Upper gastrointestinal haemorrhage (haematemesis and melaena)

Causes

Oesophageal	Gastric	Duodenal	General
1) Esophageal varices. 2) Reflex oesophagitis. 3) Mallory Weiss syndrome (severe in coordinated vomiting → longitudinal mucosal tear in lower esophagus)	1) Multiple gastric erosions. 2) Acute haemorrhagic gastritis. 3) Chronic gastric ulcer. 4) Benign or malignant neoplasms of the stomach.	1) Acute duodenal ulcers 2) Chronic duodenal ulcers	Blood diseases as: 1) purpura 2) hemophilia

The commonest 4 causes of upper GI hemorrhage are in the following order:

1. Oesophageal varices.
2. Acute gastric erosions usually caused by intake of NSAIDs.
3. Acute haemorrhagic gastritis
4. Chronic duodenal ulcer.

Management

A-Initial resuscitation

- 1- Admit to hospital (ICU in severe bleeding)
- 2- Insert two peripheral venous lines & withdraw blood for cross-matching and blood tests.
- 3- Insert a Foley catheter
- 4- IV Na containing fluids as Ringer's lactate until blood is available.
- 5- A NG tube is inserted for all cases.
- 6- Correct coagulopathy by fresh frozen plasma & by giving the missing factors as cryoprecipitate (factor VIII) for hemophilia, platelet concentrate for purpura or IV vitamin K for hypoprothrombinaemia.

-Upper GI Hge is usually due to lesions above the ligament of Treitz (end of duodenum). -It manifests by:

1. Haematemesis means vomiting of blood which may be of a coffee ground material (if small in amount) or bright red blood (if large volume)

2. Melaena

is passage of black tarry stools.

-In most cases it is caused by bleeding from the upper GI tract.

-Rarely may arise from a lesion below the ligament of Treitz, e.g., from lesions of the small bowel as neoplasms, bleeding Meckel's diverticulum, intussusception, Crohn's disease, mesenteric vascular occlusion & bleeding typhoid ulcers.

3. Fresh bleeding per rectum (If upper GI bleeding is so massive and the GIT transit is so fast

4. Occult GI bleeding (Minor trickling of blood from any part of the gut not manifest in vomitus nor change in stools color)

Urine output is the best monitor of tissue perfusion

- 7- A team approach that includes a gastroenterologist, a surgeon with expertise in GIT surgery & skilled nurses gives better results.
- 8- Endotracheal intubation → to prevent aspiration of blood (A major cause of morbidity & mortality) especially in patients with altered mental status

B-Localization of the site and cause of bleeding

I)History

- 1- Previous attacks & their management.
- 2- Hepatitis & Schistosomiasis.
- 3- Medications, particularly NSAIDs.
- 4- Peptic ulcer symptoms.
- 5- Bleeding tendency.

II)Examination

- 1-Stigmata of cirrhosis (spider angiomas, jaundice, gynaecomastia, palmar erythema, testicular atrophy, splenomegaly, ascites, cirrhotic liver)
- 2- Surgical scars.
- 3-Tenderness

III)Laboratory tests

- 1-HB % & haematocrit value will show hemodilution after **3 hours**.
- 2-Liver function tests (disturbed in patients with cirrhosis & oesophageal varices)
- 3- Blood urea & creatinine.
- 4- Coagulation tests (to exclude general causes of bleeding)

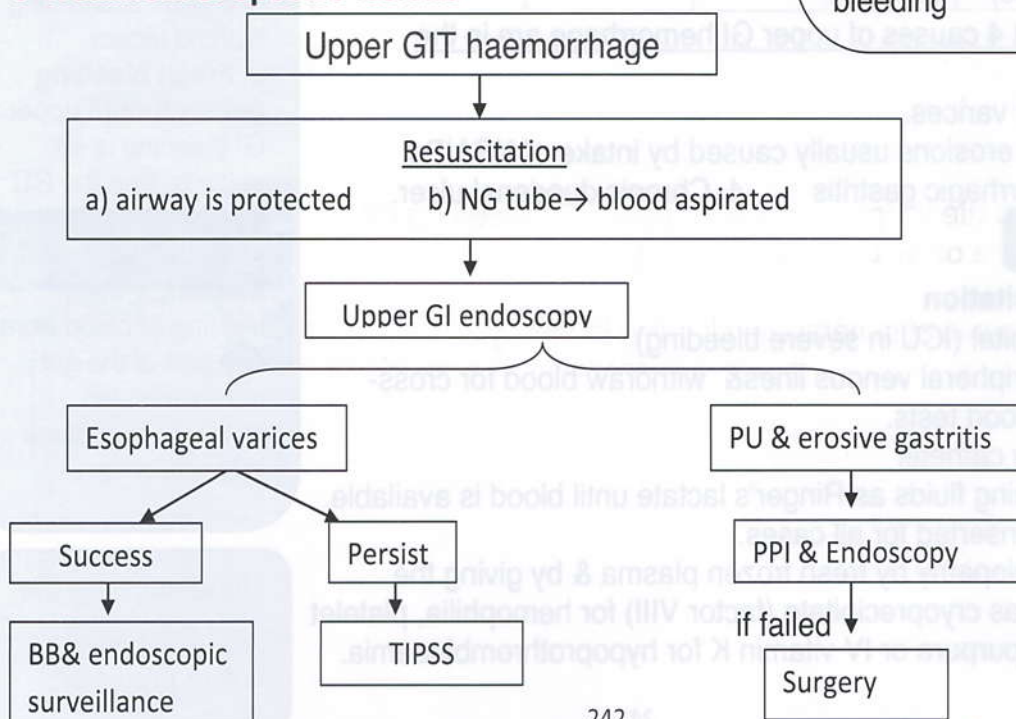
IV)Fibreoptic Endoscopy (the most important test) -

- It should be performed as early as possible once the patient has been resuscitated.
- Done under a mild sedative as diazepam.
 - Establish the source of bleeding in **90-95%**
 - It may reveal the actual bleeding spot.
 - In cases of double lesions, will tell which one is bleeding.
 - Can be used therapeutically to stop the bleeding.

V)Angiography

- Indications: difficult situations where radiology or endoscopy fails to diagnose the lesion
- Go about coeliac angiography to pinpoint the source of bleeding , e.g. angiomatous malformation of stomach.
- Performed during active bleeding

C-Treatment of specific lesions



Bleeding Per Rectum

Causes

1. **The commonest** are lesions of the anal canal as haemorrhoids and anal fissure.
2. Generalized causes: as thrombocytopenia or leukaemia.
3. Colorectal causes:

Congenital	Inflammatory	Vascular	Neoplastic
Familial polyposis coli.	1. Acute bacillary dysentery. 2. Amoebic dysentery. 3. Bilharzial colitis. 4. Ulcerative colitis. 5. Diverticular disease of colon	1. ischaemic colitis 2. Angiomatous malformation of the colon	Benign & malignant neoplasms of the colon and rectum

Clinical picture

1-Haemorrhoidal bleeding is characterized by:

- a) Fresh bright red.
 - b) Jet or drops separate from stools.
 - c) Occurs with straining usually by the end of defecation.
- 2- Patients with FPC present in 2nd decade by loose stools, attacks of lower abdominal pain, diarrhoea and passage of blood & mucous.
 - 3- In acute dysentery → abdominal pain, tenesmus & bloody diarrhoea (main symptoms)
 - 4- In ulcerative colitis → a long history of diarrhoea with rectal discharge of mucous or blood.
 - 5- Patients with ischemic colitis → usually elderly and complain of left sided abdominal pain & bright red rectal bleeding
 - 6- In carcinoma of the colon or rectum → recent change in the bowel habits, attacks of large bowel obstruction or bleeding per rectum.
 - 7- In diverticular disease & vascular malformations of the colon → clinical examination is usually irrelevant

Investigations

Check that the patient does not have upper GIT bleeding by passing a NG tube or by upper endoscopy.

A-Laboratory

1. Exclude the causes of generalized bleeding tendency.
2. Stools examination → bilharzial ova or trophozoites of amoebiasis.

B-Instrumental

1. Proctoscopy → reveal internal haemorrhoids.
2. Sigmoidoscopy → diagnose most of the lesions of the rectum, sigmoid colon & descending colon
3. Colonoscopy → visualize the whole colon

-Hematochezia is fresh bleeding per rectum.

-Causes of massive bleeding per rectum in adults:

1. Diverticula.
2. Ulcerative colitis.
3. Ischemic colitis.
4. Angiodysplasia.
5. Massive bleeding from upper GIT.

- Cause of massive bleeding per rectum in children: Meckel's diverticulum

-Facts about bleeding per rectum

1. Spontaneous remission is **80%**. (bleeding usually stops by the time the patient presents to hospital)
2. No source of bleeding can be identified in **12%**
3. Bleeding is recurrent in **25%**
4. Lower GIT bleeding is more difficult to diagnose than upper GIT bleeding

- The presence of haemorrhoids in an elderly patient does not ascertain that they are the cause of bleeding.

-Haemorrhoids & rectal carcinoma can coexist (a grave mistake to treat such a patient with haemorrhoidectomy leaving the carcinoma to advance)

C-Radiology

1. Isotope scans

- The patient's own RBCs are tagged with ^{99m}Tc then injected IV
- Abdominal scanning by a gamma camera → identify bleeding site

2. Angiography (Not easy & not available except in special centres)

- **indication:** when colonoscopy cannot be performed because of massive bleeding or when colonoscopy cannot pinpoint the source of bleeding, e.g., in angiomatous malformations of the colon.

- Method:** Selective catheterization of SMA or IMA → localize the site of bleeding (if rate of bleeding is **0.2-0.5 ml or more / minute**)

- If the source of bleeding is localized → stop the bleeding by injection of vasoconstrictors or gel foam

3. Contrast radiology.

Double contrast barium enema is only justified as an elective investigation in case of chronic blood loss.

D- Laparotomy

- Indication:** If all the previous investigations are not available or failed to localize the site of bleeding

- It may be unavoidable in patients with massive bleeding.

- Colonoscopy can be performed during exploration

Treatment

1. Fortunately in the majority of cases, bleeding will stop spontaneously → have time to diagnose and treat electively.

2. For massive bleeding → start the usual resuscitative measures → If bleeding continues → colonoscopy or angiography (according to the available experience & facilities)

- If angiography localized the bleeding point → stop bleeding by injection of vasopressin **0.2 unit/minute** or by embolization with thrombin or gel foam.

- If colonoscopy visualizes an area of vascular malformation (angiodysplasia) → stop bleeding by diathermy or laser.

4. surgical intervention

- Indications:** a) All the previous measures fail to stop bleeding
b) The bleeding is massive (**blood loss > 2,5 liters over 48 hours**)

- Benefits:** lower mortality than continued conservative management.

- Operation,** either:

- a) Segmental colon resection
(If the source of bleeding could be localized preoperatively)
- b) Total colectomy (If the source of bleeding can't be identified)

- The sigmoidoscope has 2 types:

- a) rigid → reach up to **30 cm** from the anal verge

- b) Fibreoptic → reach up to **70 cm**

- Colonoscopy:

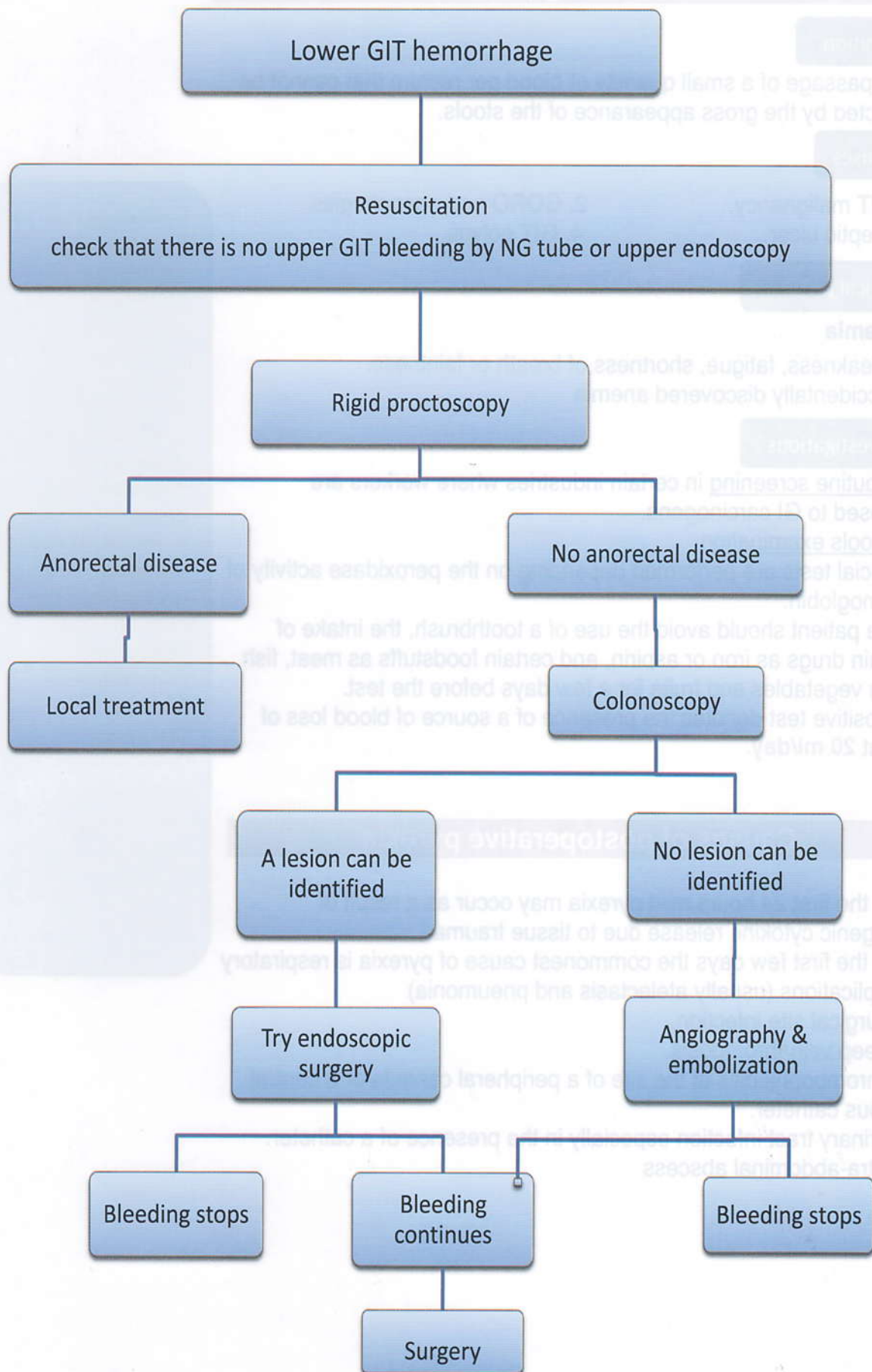
- a) needs proper preparation of colon by repeated enemas.

- b) In patients with massive colonic bleeding → blood will obscure the field (better to postpone)

- c) The investigation of choice for chronic blood loss

- Minor bleeding is treated electively (there is enough time for examination & investigations to reach a diagnosis before starting treatment)

- Massive bleeding is treated on an emergency basis as mentioned earlier for upper SI bleeding



Occult bleeding per rectum

Definition

It is passage of a small quantity of blood per rectum that cannot be detected by the gross appearance of the stools.

Causes

1. GIT malignancy.
2. GORO and oesophagitis.
3. Peptic ulcer.
4. GIT polyps

Clinical picture

Anaemia

- a. Weakness, fatigue, shortness of breath or faintness.
- b. Accidentally discovered anemia

Investigations

1. Routine screening in certain industries where workers are exposed to GI carcinogens.
2. Stools examination
 - Special tests are performed depending on the peroxidase activity of haemoglobin.
 - The patient should avoid the use of a toothbrush, the intake of certain drugs as iron or aspirin, and certain foodstuffs as meat, fish, fresh vegetables and fruits for a few days before the test.
 - A positive test denotes the presence of a source of blood loss of about **20 ml/day**.

Causes of Postoperative abdominal distension and vomiting

1. Acute gastric dilatation.
2. Paralytic ileus.
3. Intraabdominal abscess, e.g. subphrenic or pelvic.
4. Peritonitis due to a leaking anastomosis or perforation of a hollow organ.
5. Adhesive intestinal obstruction.
6. Medical causes as uraemia or diabetic keto-acidosis

Causes of postoperative pyrexia

1. In the first 24 hours mild pyrexia may occur as a result of pyrogenic cytokine release due to tissue trauma.
2. In the first few days the commonest cause of pyrexia is respiratory complications (usually atelectasis and pneumonia)
3. Surgical site infection.
4. Deep vein thrombosis.
5. Thrombophlebitis at the site of a peripheral cannula or a central venous catheter.
6. Urinary tract infection especially in the presence of a catheter.
7. Intra-abdominal abscess

Salivary gland

Congenital diseases

Sialectasis

- It is the salivary gland's analogue of bronchiectasis
- Pathogenesis: degeneration of the alveolar & duct system → become dilated → inadequate drainage → infection
- Etiology: is not known, but many cases are congenital & familial.
- Misdiagnosed in children as mumps but differ from it in being **unilateral and recurrent**.
- Treatment: Antibiotics are used during acute attacks of infection
- Usually self limited after the age of 15 years.

Infections

Viral parotitis (Mumps)

- Incidence:
 1. Decreasing in frequency because of immunization
 2. Still is common cause of salivary gland swelling
- C/P: 1. Bilateral painful parotid gland swelling
- 2. Fever
- Type of patient: children
- Treatment: the disease is self-limiting



Acute bacterial sialadenitis

Incidence

Rare in modern surgical practice

Etiology



- Causative organism: Staphylococcus aureus (**the commonest**)

-Predisposing factors:

In the past, it occurred in the elderly postoperative patients because of a combination of poor hygiene and dehydration

-Precipitating factors:

Obstruction of the Stensen's duct by a stone → infection

Clinical picture

-Symptoms:

a) **General**: fever

b) **Local**:

1. Pain: Is marked because the swollen gland lies within the tough parotid fascia.

- Increases with talking

- If an abscess forms → becomes throbbing

2. Swelling in the side of the face

-Signs:

a) **Inspection**: whole gland is diffusely enlarged & the skin is red

b) **Palpation**: 1. The swelling is markedly tender and firm

Physiology

- Saliva secreted by the parotid gland is serous fluid while that secreted by the submandibular gland has higher viscosity & rich in calcium
- Salivary secretion is continuous day & night but is increased during eating.

- Functions of the saliva

1. Food lubrication to allow swallowing.
2. Cleaning the mouth.
3. Starch digestion by the salivary amylase.
4. It mediates taste sensation.

Non-neoplastic salivary gland diseases

1. Congenital diseases: as aplasia, ectopic parotid tissue, cystic hygroma & sialectasis
2. Infections.
3. Salivary stones.
4. Salivary fistula.
5. Degenerative diseases.
6. Autoimmune salivary diseases.
7. Drug-induced, endocrine & metabolic salivary gland enlargement

2. A stone is sometimes felt in the duct by bimanual examination.
3. Gentle pressure on the gland → a bead of pus at the duct opening
4. If an abscess is formed →
 - i) No fluctuation (because the gland is covered by tough fascia)
 - ii) Known from development of skin edema



Treatment

A) Conservative management

Antibiotic → **Clindamycin** (attains the highest salivary concentration)

B) Surgical drainage

- Indications:
1. Failure of 48-hour conservative treatment
 2. Evidence of abscess formation

-Operation:

1. General anaesthesia
2. Skin incision (as that used for parotidectomy or part of it)
3. A skin flap is raised off the gland
4. A mosquito forceps is thrust in the deep fascia at the suspected area then gently opened along the direction of the fascial nerve branches to avoid their injury (**Hilton's method**).
5. Pus is sampled for culture and sensitivity.
6. A rubber drain is inserted for a few days till discharge stops.

Salivary stones (sialolithiasis)

Incidence

Submandibular > parotid and sublingual glands
(As the saliva of the submandibular gland is viscous and has the highest calcium concentration)

Pathology

- Stones sometimes form from the constituents of saliva.
- Stones lead to chronic and recurrent sialadenitis.
- Sjogren's syndrome → reduced saliva flow → stones formation
- The stone either stays in the gland or in the duct.

Clinical picture

1. Painful gland swelling while eating (stones block salivary ducts)
2. Acute or recurrent attacks of sialadenitis

Investigations

1- Plain x-ray (**occlusal view**)

A stone in the submandibular gland is radio-opaque in **80%** of cases
(Because of its high calcium content)

2. Ultrasound: show almost all stone



Recurrent subacute and chronic sialadenitis

- Etiology: 2^{ry} to an abnormality in the salivary gland as: Sialectasis, stones, or autoimmune diseases

- C/P of chronic sialadenitis of the submandibular salivary gland: a swelling in the digastric triangle which may be confused with submandibular lymph nodes.

- The disease is characterized by:
1. History of pain and increase in the size of the swelling during eating.
 2. The swelling is solitary and cannot be rolled over the mandibular edge
 3. Bimanual palpation → the swelling fills the floor of the mouth.



Treatment

- Surgical (according to the site)
 - a) A stone palpable within a duct → removed through the mouth
 - b) Stone within the gland substance → removed with the submandibular or parotid gland.

- Parotidectomy for calculi disease needs high experience as the recurrent inflammation leads to difficult facial nerve identification

Degenerative diseases

Sialectasis

Definition

Abnormal dilatation of the small branches of salivary ducts & alveoli

Etiology

The cause is not known but the childhood type is known to be a familial disease.

Clinical picture

Recurrent attacks of sialadenitis

Investigations

- Sialography (the investigation of choice)

-Technique: 1. The salivary duct is cannulated and a contrast material is injected into the duct

2. Films are taken before and after the patient sucks a lemon for several minutes (**Postevacuation film**)

-Findings: 1. The degenerating alveoli coalesce and become cystic → **snow storm appearance**

2. Punctate sialectasis may be seen in cases of Sjogren's syndrome
3. The ducts show alternating strictures and dilatation

Treatment

A) Conservative treatment

1. Finish every meal by a citrus drink → Stimulate salivary flow then massage the affected gland → to squeeze out the accumulating epithelial debris.
2. Antibiotics as clindamycin (treat infection)

B) Surgical excision (rarely needed)



Radiation sialadenitis

-Etiology:

radiation to the nasopharynx or the skull base.

-C/P: Salivary secretion is temporarily suppressed

-Treatment: administration of sialagogues as citrus fruits

Autoimmune salivary diseases

Types

A) Sjogren's disease

-Incidence: commoner in **women** than men.

-Clinical picture: 1. dryness of the mouth (**xerostomia**)

2. Dryness of the eye (**xerophthalmia**, **keratoconjunctivitis sicca**)

3. Rheumatoid arthritis.

4. Sialomegaly or salivary gland discomfort (in some patients)

- Etiology: 1. Not exactly known
- 2. May be due to CMV → make the salivary glands ducts antigenic → infiltration of the glands with lymphocytes around the ducts → ducts obliteration

B) Benign lymphoepithelial lesions.

- Incidence: uncommon disease
- Pathology: progressive lymphocytic infiltration → diffuse salivary glands enlargement (particularly submandibular and parotid)
- Type of patient: common in middle aged and elderly women.
- Prognosis:
Has prelymphomatous potential (the term benign is misleading)

Complications 20% of cases develop lymphomas.

Diagnosis

- 1-Lip biopsy: includes the minor salivary glands
- 2- Parotid sialography, shows:
 - a) nonspecific appearance of narrowed ducts
 - b) punctate sialectasis (sometimes)

Treatment

- 1- Artificial tears made up of **methylcellulose** → reduce eye dryness
- 2- Careful oral hygiene.
- 3- If patient develops a palpable mass in parotid → do needle biopsy
- If this proves to be a lymphoma, the gland is removed, the disease staged & treatment is initiated with radio and/or chemotherapy.

Salivary Neoplasms

Incidence 1.2% of all neoplasms & 5% of head and neck tumours

Pathology

- The majority is benign and most commonly arise in the parotid
- The incidence of malignancy varies inversely with the size of the gland → Malignancy occurs in:
 - 25%** of parotid neoplasms
 - 40%** of submandibular neoplasms
 - 70%** of sublingual & minor salivary
- Origin: the secretory tissue, the duct system or lymphoid tissue

Types and classification

Benign	Malignant
1. Pleomorphic adenoma	1. Mucoepidermoid carcinoma
2. Adenolymphoma (Warthin's tumour)	2. Adenoid cystic carcinoma
3. Oncocytoma (oxyphil adenoma)	3. Acinic cell carcinoma
4. Monomorphic adenoma	4. Adenocarcinoma
	5. Carcinoma ex pleomorphic adenoma

- Patients with Sjogren's disease has alteration in the relation Ship between T-helper and T-suppressor cells → 44 times prone to the development of lymphoma than the general population.

- Drug-induced, metabolic and endocrine salivary gland enlargement

1. Drug-induced enlargement has been reported with:
sulfisoxazole, phenylbutazone, iodide containing compounds, thiouracils, hypotensive drugs, Contraceptive pills

2. Metabolic and endocrine causes include: liver cirrhosis, DM, Alcoholism, malnutrition & ovarian, thyroid or pancreatic insufficiency.

Benign neoplasms

Pleomorphic adenomas

(The commonest salivary tumour)

-Incidence: 1) **75%** of parotid & **50%** of submandibular gland neoplasms

2) Equal in both males & females

-Macroscopic picture: characterized by an incomplete capsule that allows extension of the neoplastic epithelium into the surrounding tissues

-Microscopic picture:

1. Has epithelial, myoepithelial and stromal components

2. Has wide variations in cellular and architectural morphology.

-Course: 1. It grows slowly without infiltrating the facial nerve.

2. Long-standing (more than 10 years)

3. Rarely turn into carcinoma

Adenolymphoma (Warthin's tumour)

-Site: usually in the lower part of the parotid gland

- Bilateral in 10 -15% of cases.

- Incidence: 15% of all parotid tumours

-Origin: arise from heterotopic salivary tissue in the parotid LN.

-Microscopically: formed of epithelial lined spaces filled with creamy material, that are surrounded by lymphoid tissue

-Consistency: Soft or cystic

-Type of patient: elderly people

-Predisposing factor: smoking

-Does not turn malignant

-Indications for operation:

1. Cosmetic purpose
2. Unsure of diagnosis

Malignant neoplasms

Adenoid cystic carcinoma

- The commonest malignancy affecting the minor salivary glands.

-Its distinctive features are slow rate of growth & perineural spread → infiltrates for a long distance in the perineural tissues of adjacent nerves → incompletely removed → high recurrence rate

Mucoepidermoid carcinoma

- The commonest type

-Origin: arises from the duct epithelium

-Site: usually the parotid

- Grades: **3**

1) Low (the most common & can affect children)

2) Intermediate

3) high- grade tumours.

Differential diagnosis

A) Extra parotid swellings

1-LNs (parotid or upper deep cervical)

2- Lipomas (mimic pleomorphic adenomas)

3- Mandibular and maxillary tumours (mimic parotid enlargement)

4-Hypertrophy of the masseter (bilateral in most cases)

B) True parotid enlargement

-Caused by early mentioned non-neoplastic salivary gland diseases

-The gland is diffusely enlarged with no definite lump.

Hypertrophy of the masseter

-Etiology: in women with habitual involuntary grinding of their teeth or in those who have

Orthodontic treatment

-Investigation: CT scan in difficult cases to differentiate it from a true parotid enlargement

	Benign neoplasms	Malignant neoplasms
Symptoms	Swelling on the side of the face which is: 1. Painless 2. Present for months or years 3. Slowly growing or has stopped growing.	A) Swelling on the side of Face that is: 1. Steadily enlarging 2. Sometimes painful ,radiating to the ear & increases with mastication B) In carcinoma ex pleomorphic adenoma → patient may give a history of a painless swelling that has been stationary for years & is now getting bigger
Signs	A) Swelling that is: 1. Localized to the parotid 2. Hemispherical in shape 3. Raises the ear lobule 4. It may attain a large size 5. Non tender 6. Firm in consistency (an adenolymphoma feels soft or cystic) 7. Smooth or bosselated surface. 8. Does not infiltrate the skin, the masseter nor mandible B) Examine the mouth cavity as tumours arising in the deep part of the parotid may bulge in the oropharynx behind the tonsil	A mass that is: 1. Usually warm 2. Mildly tender 3. Firm or hard and has an Irregular surface. 4. May be adherent either to the skin, the masseter or the mandible
Facial nerve infiltration	Not present	Present with evident weakness or paralysis of facial muscles
Cervical LNs	- Not enlarged - No evidence of distant metastases	- Sometimes involved. - Rarely metastasize to the lungs

Salivary malignancies do not usually grow as fast as other cancers in the body



Pleomorphic adenoma



Malignant parotid tumour with left

Investigations

A) Pathological diagnosis

-Indication: before operating on tumours that are clinically malignant

-Types:

1- **FNAC** (Fine needle aspiration cytology)

-Is reliable in the hands of an expert cytologist

2- **Excision biopsy**

-For tumours of the minor salivary glands of the mouth cavity

B) Imaging

1- **CT scan and MRI** (the most useful)

2- **Isotope scan with technetium**

(**Tc99m**) **pertechnetate** Adenolymphoma

and oncocytoma concentrate technetium

→ show as hot spots



Treatment

-By Surgery (the only reliable form of treatment)

A) Clinically benign tumours

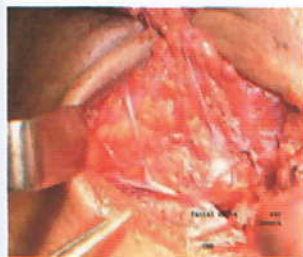
According to the site of the tumor:

I) In parotid tumors →

Superficial parotidectomy (the standard treatment)

-Operation:

1. Removal of all parotid tissue that is superficial to the facial nerve and its branches (taking great care not to injure them)
2. Ensures removal of the tumour and its tiny tissue extensions outside the defective capsule.
3. Simple enucleation is contraindicated → leads to high incidence of recurrence
4. If nerve is accidentally injured → immediate repair by microsurgical techniques either by direct suturing or by a nerve graft from the great auricular nerve.



II) In submandibular gland tumours →

Submandibular sialadenectomy

-Operation:

1. An incision is made parallel to & 2 cm from the lower border of the mandible (to ensure safety of the mandibular division of facial nerve)
2. Lingual & hypoglossal nerves are at risk when dissecting the deep aspect of the gland (should be preserved)

Diagnosis of salivary gland lumps is clinical, in a minority of cases investigations are needed

- Open surgical biopsy of the major salivary glands is contraindicated, FNAC is safer

- Surgery for malignant tumors is extensive & mutilating as it may entail sacrifice of the facial nerve so one should be backed with a pathological proof of malignancy before operation

- Patient should be warned against the possibility of accidental facial nerve injury or intentional sacrifice (in malignant lesion)

- Facial nerve weakness occurs even with successful preservation (because of neurapraxia) that (recovers after a few months)

- The majority of parotid pleomorphic adenomas arise in the part that is superficial to the facial nerve



B) Clinically malignant Tumours

I) Radical excision (The standard treatment)

-Operation:

1. Wide surgical clearance with cervical LN dissection if they were enlarged
2. For the parotid → excision of the facial nerve and probably part of the masseter or the mandible.
3. Treatment is modified according to aggressiveness of the tumour.

II) Radiotherapy

- Is of limited value but is administered as a postoperative adjuvant therapy for tumours of high-grade malignancy.



- If a pathological diagnosis has not been obtained prior to the operation, frozen section examination during surgery is helpful

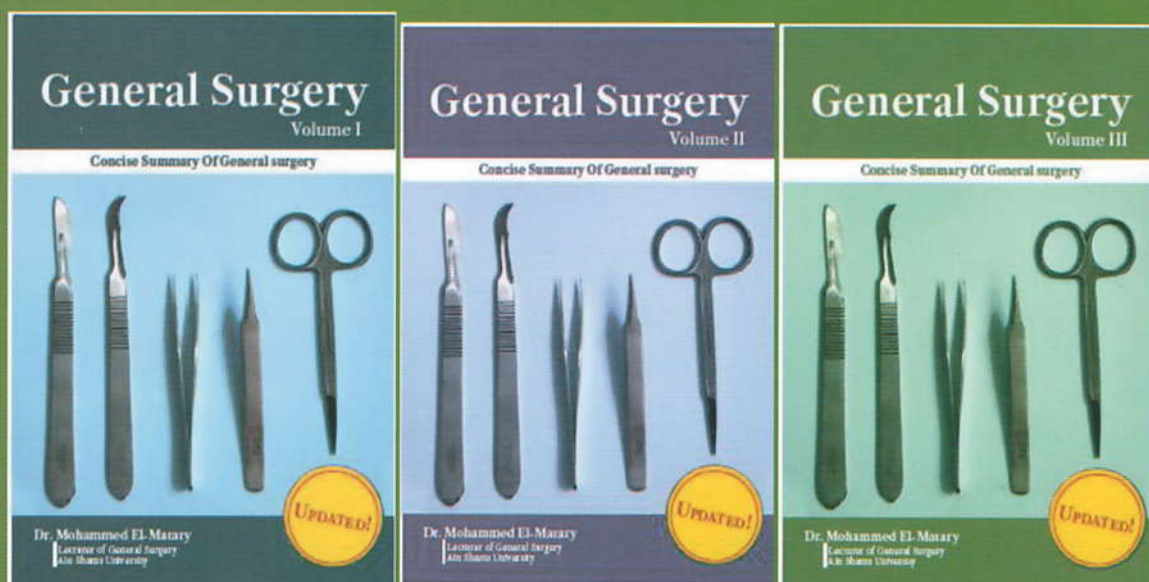
- For the low grade mucoepidermoid carcinoma, an attempt to preserve the facial nerve is secured

- Parotid pleomorphic adenoma is important because:

1. Very near to facial nerve which may be damaged at surgery for its removal
2. If the capsule is damaged during removal or if it were just enucleated → high recurrence rate
3. Long standing tumors may turn malignant

- Malignant parotid tumors are suspected with:

1. Short duration and progressive growth
2. Pain or tenderness
3. In advanced cases → classical signs of facial palsy and LN enlargement



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